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*Deceased.

Editorials

THE PLATFORM OF THE AMERICAN MEDICAL ASSOCIATION

The American Medical Association advocates:

1. The establishment of an agency of the federal government under which shall be coordinoted and administered all medical and health functions of the federal government exclusive of those of the Army and Navy.

2. The allotment of such funds as the Congress may make available to any state in actual need, for the prevention of disease, the promotion of health and the care of the sick on proof of such need.

3. The principle that the care of the public health and the provision of medical service to the sick is primarily a local responsibility.

4. The development of a mechanism for meeting the needs of expansion of preventive medical services with local determination of needs and local control of administration.

5. The extension of medical care for the indigent and the medically indigent with local determination of needs and local control of administration.

6. In the extension of medical services to all the people, the utmost utilization of qualified medical and hospital facilities already established.

7. The continued development of the private practice of medicine subject to such changes as may be necessary to maintain the quality of medical services and to increase their availability.

8. Expansion of public health and medical service consistent with the American system of democracy.

THE POLITICAL DOCTORS

The desire to heal and cure is one of the most abiding compulsions of the human race. Comparatively few will wish to build or repair a machine, appear at law, teach, or lay bricks but practically everyone will at some time give advice on or actually perform, under his own direction,

the opening of a pimple, the taking of aspirin, the planning of a reducing, gaining, or strengthening diet or the prescribing of a cold cure or a laxative. Practiced in this way the ancient compulsion has had its satisfactions and its tragedies. Some have had aspirin for diphtheria and others cathartics for appendicitis. The mistakes have of course stemmed from folk lore which at one point precedes but eventually falls behind the accumulations of diagnostic and therapeutic information which comprise a science.

The desire to heal and cure is not confined to contacts with the individual but is demonstrated in religious and other social practices. Of these practices religion is the oldest by far. Compared with younger systems of dealing with social ills religion is the only one with a mature body of philosophical and practical concepts. The very maturity of religion removes from it the romantic halo which surrounds social welfare. For instance everyone knows that to make religion effective work is required—individual labor and self-denial whereas the outstanding proponents of current social welfare extension appear to believe that the ills of society can be relieved by legislative acts and the expenditure of great sums of money. These romanticists, the social welfare group, have gone about their urge to cure by seeking to work on a very big and most dramatic patient—the body politic. And since everyone wants to do some curing they have been quite successful in getting the permission and assistance of legislators and executives.

The greatest set of diagnoses, without successful treatment, has been prepared for the American body politic. Its woes are economic, therefore, its economy must be changed. Its agriculture needed adjusting because it was hyperfunctioning. Part of it was exercise. The doctors consulted once more. The banking was hypofunctioning. A few ramifications of this system were trimmed off and a series of injections—substitutive therapy—were administered. The altered and inactive financial glands just laid down and waited. The industrial system wouldn't work hard enough in some parts. So the parts that would work had a lot of their excess and a bit of their reserves taxed off to keep the rest of it going by passive exercise. But passive exercise never restored a dwindling nerve and the industrial system just went along with odd shapes of hypertrophy and atrophy.

Having proclaimed a set of diagnoses of the economic body thereby keeping the physical body distracted although never quite having the fortitude to openly face the diagnosis and the limitations of treatment the great doctors went on to inspect, pummel and percuss the American body's completely physiological requirements.

Overlooking its established physicians' inheritance and their sponsorship and practice of public health measures and their prevention of disease through public and private education it was decided that these old docs 1, charged too much; 2, didn't see the sick poor; 3, or, if they did, took better care of them than of the medium income group; 4, couldn't properly care for the poor under locally supervised and well-established controlled expense methods, and 5, didn't have enough hospitals (although hospitals were reasonably close to everyone).

At this point the great doctors' diagnoses came to resemble exactly a set of European specialists' opinions. Looking back one could see that the treatment outline had followed the European plan too. But the Europeans' health hadn't been superior to the Americans'. They had changed only by becoming wards of the paternalistic State. It seems likely that the psychological health of such peoples would eventually be impaired for paternalism past a youthful age can not help but interfere with mature development.

The federal government, and to a lesser extent other divisions of government, in the last several years has projected itself into medical practice in such manners as to cause all thinking physicians to evaluate anew the relationship of socially minded legislative performers to the field of medical practice. Which is to say that since politics has intruded without invitation into medicine, medicine will have to nose into politics. The physicians of Illinois must decide to what extent their societies and publications shall feel free to point out that *certain individuals in politics detrimentally effect the cure of the sick.* In so doing we will abandon our historic neutrality in politics but the time has come to be for or against candidates for office because of their views or practices affecting our well thought out, effective and time-tried methods of caring for the ill. We are counselled by many of our colleagues to make no overt move—the powerful

will be offended and move against us with *vengeance.* We are fighting the social philosophies of a coterie influencing national executive thought and legislation. Appeasement is absurd.

HEALTH PROBLEMS IN EARLY CHICAGO

Two of the most pressing problems in connection with public health in Chicago from 1840 through the sixties were the control of contagious disease and the collection of vital statistics through adequate provision and enforcement of death registration.

As early as May 10, 1841, the Common Council concerned itself with vital statistics. An ordinance, which appeared on June 25 in one of the daily papers, made it obligatory for the attending physician to issue a certificate to the head of the family of a deceased patient.

The certificate was to specify age, sex and cause of death, whether by accident or disease, and the nature thereof. However, it made no provision for securing addresses or the location of deaths as to neighborhoods. A fine of $5 was imposed on the physician for failure to comply with the ordinance.

The head of the family, in turn, was to be responsible for the deposit of the certificate with the City Sexton previous to interment of the deceased, under penalty of $2 for each omission.

With no adequate supervision to see that the ordinance was carried out, it is no wonder that little was done. On February 27, 1841, an amendment to the City Charter provided for the election of a City Marshall, who later on frequently assumed the duties of Health Officer, though during the early forties he did not act as such.

There is no record of deaths whatsoever previous to 1843, but in that year an effort to provide vital statistics was made by the General Assembly of Illinois, which in April passed an Act to establish a mode of registering births and deaths with the clerk of the County Commissioner's Court. Parents or any householder might make the affidavit of birth, giving the Christian name, day and year, and "Justice's precinct." The eldest relative or executor of a deceased person was to register the name and age, as well as the "precinct" where the death occurred.

However, statistics for the following years were unreliable and, such as they were, were gathered by the Board of Health of 1869 from newspapers, medical journals, undertakers' and sextions' reports.

The first year in which a record of deaths was kept by the City Sexton was 1847, and these records were inaccurate. Difficulty in estimating mortality figures in 1850 was shown by some old documents which have been preserved. These were mere scraps of paper from doctors, sextons and the Board of Health. In the case of burials it was impossible to tell what dates were covered. The list of deaths were frequently in pencil, some giving no names, and but one giving the cause of death.

The first record, prepared by the City Clerk from the records of the City Sexton, which gave the names, ages and causes of death of those buried in the city, was dated June 1, 1851. Later in the year another ordinance required the attending physicians to convey the certificate of death to the Sexton before burial, instead of leaving it to a member of the family. Moreover, the City Sexton's record was to state the name, age, cause and date of death, section where the death occurred, and if obtainable, place of birth.

There were two flaws in these requirements: first, the report did not include persons sent elsewhere for burial; second, the causes of death listed were far from enlightening, "unknown" being one of the most frequent. Others included such diversified causes as "decline," "visit of Providence," "want of breath," "never was well," "heat in brain," "effects of sea voyage," and "hereditary complaint."

From May until December, 1854, there were many deaths from cholera, but during the same year four hundred and seventy-eight deaths were reported from "unknown causes," and one hundred and seventy-nine without any name.

Perhaps one explanation for laxness in securing complete death registrtions lies in the method of compensating the City Sexton, whose livelihood depended solely on the sale of lots in the City Cemetery. No wonder his reports were inaccurate!

The effort to insure registration of deaths was continued, but with little success, throughout 1861. Due to many burials in other places than the City Cemeteries, as well as outside the

city limits, complete statistics were almost unavailable.

The Chicago Medical Society was largely responsible for the attempts made after 1861 to remedy the situation. In January, 1864, the Society appointed two physicians to present its views to the health officials. They asked that "a method of tabulation be adopted calculated to set forth distinctly every important fact contained in the books of registration." In accordance with this request the City Attorney prepared an ordinance, which was introduced into the Council. No action was taken however.

On November 20 and 27, 1865, the Common Council considered the matter of vital statistics once more, this time in connection with the establishment of a Board of Public Works, whose duties were to be to clean up the streets and arrange for the collection of garbage.

This ordinance required every practicing physician who had "any patient with infectious or pestilential disease" to send a report to the Secretary of the Board of Police, and in case the patient died, to send the Secretary a certificate "giving age, sex, residence and cause of death." The friends of the decreased were to have a copy to show to the Sexton. The latter was required to inscribe on the back "his name, day of the month and the year." A fine of $50 for each offense was to be imposed on the physician or Sexton in case of refusal or neglect. These certificates were to be filed and tabulated for publication every week by the City Physician.

The Chicago Medical Society had sponsored the ordinance, and its provisions were to be carried out under a consulting board of physicians, to be appointed by the Society, who had offered to serve without pay. The minutes of the Council Meeting for November 24 recorded that the City Fathers asked the Society to appoint a committee of five to confer with them in regard to measures to prevent cholera during the coming year.

Before the meeting on November 27 the Society addressed a document to the Mayor and Council which set forth its program for the protection of public health. One of the points made was rigid enforcement of certificates as to the cause of death. The doctors pointed out the need of proper tabulation and the value of knowing the exact location of deaths in order to trace sources of disease. They called the contemporary registry a "ridiculous and shameful farce."

In spite of the fact that the Council passed the ordinance on November 27, the Mayor vetoed it. Possibly he resented the Chicago Medical Society's direct approach to the Council and its apparent disregard of the Board of Police; on the other hand, the Society probably objected to the transfer of health matters to the Police Commissioners.

The Chicago Medical Society, represented by its committee, came before the Council again on May 7, 1866, with a petition for a sound registration of vital statistics. Curiously, the Council referred this to the Committee on Wharves and Public Grounds, who on June 8 presented the ordinance drawn up in March, 1864, providing that a physician tabulate the mortality returns. The Council refused to consider this plan.

On July 23 the ordinance was again presented, with the amendment that the Health Officer or Secretary of the Board of Police tabulate the returns under the direction of the City Physician. It further provided that the tabulation show the number of deaths in each division of the city, by months; the sex, color and nativity of each person; the number of deaths of children under five; and the cause of death.

However, the registration was still in the hands of the inadequate officials, and no improvement made. The members of the Chicago Medical Society had been willing to give their services, and it had even been pointed out in one of the Society meetings that it was the duty of the profession to instruct the city authorities in their duties. Unfortunately, cooperation could not be obtained at this time. Consequently vital statistics remained unreliable in spite of valiant efforts put forth by the medical profession.

It was on July 1, 1867, that the Medical Society finally got an ordinance passed by the City Council which resulted in the return of the first accurate mortality records for Chicago in 1868. This ordinance required registration of all births and deaths with the Board of Health, in case of the latter within thirty-six hours. Besides the coroner was to make a written statement after every inquest as to the cause, date and place of death.

Even more difficulty was experienced in controlling contagious disease in the early days of

Chicago than in securing vital statistics. In 1839 there had been legal provision for a health program, followed by sufficient epidemics with which to cope, but the Board of Health met only sporadically and the duties of health officer frequently fell to the City Marshall. Upon the dissolution of the Board in 1860, the Police Department took over its functions.

In 1843 general fear of cholera contagion prompted the erection of the first hospital for contagious diseases near Lake Michigan at the foot of North Avenue, which at that time was far beyond the city limits, on land purchased for a cemetery. It was a rude board structure costing but $200.

The early shelter burned to the ground in 1845; however it was soon rebuilt and then became known as the "Pest House." In the meantime patients were taken to the house of a Mr. Shaw, which was located in the northeastern port of the city.

Dr. James V. Z. Blaney mentioned an epidemic of scarlet fever in 1844 and also referred to the prevalence of diarrhea and disentery. The Board of Health apparently was inadequate to control the citizenry and impose obedience to its orders, for in 1845 it asked the Common Council by what authority it could abate nuisances when no attention was paid to its demands. The Council evidently did nothing about the matter.

In 1846 for the first time the same man was called City Marshall and Health Officer.

The following year, the same that saw the establishment of the County Hospital, another smallpox scare occurred. Immediately the *Democrat* hastened to reassure newcomers that "although this disease has several times made its appearance in our midst, it has never been allowed to spread beyond first cases."

A smallpox epidemic actually did strike the city in 1848. On January 28th the City Marshall advertised the names of physicians who would vaccinate the poor free of charge. A. T. Andreas states that "this appears to be the first official and medical co-operation for precautionary measures to prevent the intoduction or spread of smallpox."

During the same year a City Physician was appointed. He served without salary and this duties were two, to publish in English and German on handbills the names of doctors willing to vaccinate gratuitiously; and to call on unvaccinated persons, give them a handbill, and advise immediate vaccination. In addition there was a tightening of quarantine regulations for immigrants.

An ordinance officially recognizing the City Marshall as Health Officer was passed on March 9, 1848. He was to "cause the removal or abatement of all nuisances which may come to his knowledge. To remove to the Hospital all persons afflicted with contagious or pestilential diseases, under the direction of the Board of Health, or the Mayor of the City; to cause the same to be suitably provided for, to keep a strict acaccount of all expenditures for said purposes and report the same, together with the vouchers therefor, to the Common Council for payment." For his services as health officer, he was to receive the munificent sum of $75 per year.

The expected advent of Asiatic cholera caused the direction of public attention to cleaning up the city streets as a preventive measure. On April 2, 1849, the Council appointed twenty-eight Assistant Health Officers to aid in this task. Three weeks later, seventeen more assistants were named.

Cholera was brought to Chicago on the immigrant ship "John Drew"; the new city did not wish to frighten away immigration, so the Mayor and Board of Health apparently tried to minimize the danger, for a newspaper article accuses the Board of Health of not reporting the "alleged cases of cholera." Finally the Common Council empowered the Board of Health to build a temporary hospital for indigent persons attacked by cholera. This structure was located on 18th Street near the river.

Unfortunately, no quarantine was established when on June 12th of that year the presence of cholera in the city had to be openly admitted.

Another shelter for cholera patients, located on Erie Street between Wells and the River, was 16 feet wide, 40 feet long and 12 feet high. It was divided into three rooms and was to be used for homeless and friendless persons. Twice during its existence it was threatened with demolishment. Closed in September, 1849, it was reopened after the "Nile" brought in a number of Swedish immigrants ill with cholera. The first private hospital, built in 1850, was likewise of a temporary nature.

Although there had been permissive legislation

to set up quarantine regulations since 1835, the only gesture in this direction in 1849 was made by the Board of Health on Aug. 13, when it recommended that the Common Council consider immediately taking measures to establish a quarantine for the city. It seems that the recommendation went no further than referral to a committee, since the city did not set up a definite quarantine until 1854.

From 1851 to 1860 there was a gradual dissolution of the Board of Health. A revision of the City Charter in 1851 did, however, require the health officer to report infectious diseases, and for the first time provided for placarding houses in which there was smallpox, as well as introducing compulsory vaccination.

Detailed provisions for quarantine stations were also made, and the Mayor was authorized to issue a proclamation requiring all public conveyances bound for Chicago to stop at such stations for the discharge of infected passengers or things. One or more physicians were to go on board to determine who and what should be stopped and attend those quarantined. The Mayor and Board of Health might also refuse to admit emigrants into any or all of the stations. In order to enforce these regulations the Mayor was empowered to keep a police force at the quarantine stations, and provision made for a fine of from $2 to $100.

When the Pest House was reopened in May, 1852, smallpox and cholera patients were separated; in addition to a hospital shanty was built "on the beach in the North Division." The following year smallpox was so prevalent that the Pest House was removed to a spot farther from the populated section of the city.

Finally, on July 3, 1854, the Council passed an ordinance which established quarantine grounds on the main lines of emigrant travel and near the city hospital. Recommendations were made to the Board of Health that the poor suffering from contagious diseases should not be sent to the Poor House to infect others.

In April of 1855 the award of $100 for the best plan for a permanent city hospital was made, a step in the direction of providing more than a series of shanties for patients with contagious diseases. In November of the same year plans were drawn for a smallpox hospital, which was constructed "upon the southeast corner of the cemetery grounds," in what is now Lincoln Park. It accommodated twelve patients, and those too poor to afford a physician.

As soon as cholera and smallpox died down, so did interest in health conditions. The result was that from 1856 until its dissolution in 1860 the Board of Health was inactive. From the spring of 1860 to 1867 health matters were entirely in the hands of the police. When the office of City Physicians was abolished in 1860 no provision was made for the duties which he formerly performed, while matters of health were referred to the Street Commissioners.

This situation prevailed until the Mayor appointed Charles S. Perry as Acting Health Officer in 1862. He caused the Pest House to be reopened, but without a City Physician there was no one to care for the patients there or those in their own homes who could not afford medical attention. The need was so crying that a City Physician was appointed later in the year at the meager salary of $600 a year. In addition he was expected to furnish his own medicine. Since he was not required to vaccinate, smallpox increased. The Pest House proved too small, and the Common Council ordered a new one, but did not appropriate funds to provide it. Conditions were made worse by the fact that the military authorities took over the general hospital, which caused removal of the contagious patients to the Poor House at Jefferson.

The next couple of years under police jurisdiction showed a decline in diphtheria and the disappearance of scarlet fever, but smallpox trebled, erysipelas nearly doubled and measles were five times as prevalent.

Cholera warnings were published in 1865, and in 1866 a new epidemic occurred. This time more stringent measures were taken. The Board of Police hired a thousand men to help clean the city, and the City Physician was ordered to place doctors on incoming trains at a point ten miles from the city to examine passengers. He was also to place health officials at depots to assist with any patients found. These precautions persisted for but three months, but it was the fear of cholera epidemics which gave birth to plans for the reëstablishment of a Board of Health.

Improvement in health matters was in large part due to the efforts of the Chicago Medical Society. It was partially responsible for the taking over of the general hospital by the County.

Its urging and the latest epidemic of cholera were the stimuli which in the end caused Chicago to take control of health matters from the Police Department and create a new and adequate Board of Health in 1867. The Chicago Medical Society had an important part in drafting the new ordinance which was responsible for its existence. Three physicians were to serve with the incumbent health commissioners and one clerk was to be appointed to keep a perfect record of vital statistics.

One civic-minded citizen went to Boston, Philadelphia and New York to learn how these cities managed public health. The committee which drew up the bill for the legislature adapted to its needs the New York plan of a metropolitan board of health. Opposition arose in the Common Council, which wished to continue the system of Police Control. However this was overruled and the Board of Health officially created by the Legislature on March 9, 1867.

Contagious disease control and the collection of vital statistics entered a new era under the recreated Board of Health. Although they still remained important items to be dealth with, efficiency in handling them increased many fold.

BE PREPARED FOR WAR INCOME TAX

Estate and Tax News releases the following information: "Some idea of the income tax that individuals will be required to pay in event of war may be gained from a study of the Connally bill. This bill, although eliminated from the final 1940 Revenue Act by the Conference Committee, was added to the Senate version of that Act by a vote of 51-28, and at least as late as June had full Treasury support. Moreover, in essentially the same form the bill at one time passed the House.

If this bill should be enacted into law, immediately upon the outbreak of war: Normal tax would be increased from 4 to 10 per cent.

Surtax rates would start at 6 per cent. on "surtax net income" in excess of $1,000 and not in excess of $2,000 (surtax now starts at 4 per cent. on surtax net income in excess of $4,000). On all surtax net income over $50,000 the surtax rate would be 80 per cent.

Exemption for married persons and heads of families would be reduced to $1,600 (now $2,000). Single individuals' exemption of $800 would remain the same. However, existing 10 per cent. earned income credit would be eliminated. Credit for dependents would be cut from $400 to $250.

Here is an example of the effect of the war rates. Assume a married man, with two dependents. Net income is $10,000, all from personal earnings.

Under the 1939 law he would have paid a tax of $343.

Under the 1940 law, his tax will be $440.

Under the Connally war tax law, his tax would be $1,702—almost five times the 1939 tax.

"MEDICAL PREPAREDNESS"

Among the most important considerations in preparation for modern warfare is that of medical defense. Physicians and surgeons have many duties in modern armies in the field, in first-aid stations, and in base hospitals. Then there are many places at home where proper medical and surgical care must be considered as essential in both preparation and defense. Medical care of the civilian groups, and the highly important care of those industrial workers engaged in the manufacture of war materials, must all be given proper consideration.

It is generally admitted in this country that the United States is not engaged in war and it is generally believed that full preparedness is the best method of keeping out of war. At this time the subject of medical preparedness is one of the most important issues before the medical profession in America, and this will be considered as a major necessity in all plans for national defense. At the annual meeting of the American Medical Association recently in New York City much of the time of the House of Delegates was utilized for the consideration of plans for medical preparedness.

The American Medical Association Committee on Medical Preparedness was created through proper action taken by the House; the Committee consists of ten physicians selected from all parts of the United States. A questionnaire form has been sent to every physician in the United States which he is expected to fill out and return to the A. M. A. committee officials whose address is in care of the American Medical

Association, 535 North Dearborn Street, Chicago.

Some physicians who are members of military or naval units, or members of the Medical Reserve, have had the impression that it is not necessary for them to submit their questionnaires, but every physician regardless of age, physical condition or membership in any organization should complete the questionnaire and return it as quickly as possible.

The A. M. A. Committee has selected a chairman for each state and territorial possession of the United States who may be considered a liaison official between the state medical society and the general committee. A state committee has been formed consisting of the officers of the state medical society, the state chairman, and the representatives from that zone area on the A. M. A. committee. Each county medical society is likewise expected to form a Committee on Medical Preparedness within the county society to aid the state committee in the various duties assigned to them.

Thus it can be seen that the medical profession is setting up its own organization for national defense as its contribution to any proposed preparedness movement. During the World War there were approximately 33,000 physicians in the various branches of service in addition to some 3,000 who were participating in other essential service for the Government. If the United States should again extend a call for medical volunteers, the response will no doubt be as gratifying as it has been in the past.

As the first essential duty it is the intention of the Committee on Medical Preparedness to provide the necessary information to the Government relative to the physicians of our country as a part of its duties in the campaign for preparedness. It is, of course, generally conceded that there is less danger of actual war if our nation is adequately prepared.

We have been informed that a few physicians in Illinois have failed to receive the questionnaire and we will be anxious to supply a duplicate letter and questionnaire to all who failed to receive the first one, or to any who have misplaced them. It is highly desirable, however, that no physician submit a second questionnaire as this would cause much confusion at A. M. A. headquarters where the data are being transferred to punch cards for future reference.

The Illinois State Medical Society Committee on Medical Preparedness is composed of Dr. J. S. Templeton, President; Dr. L. E. Day, Chairman of the Council, and Dr. Harold M. Camp, Secretary and also state Chairman for the A. M. A. Committee. It is the desire of our State Committee to send frequent informative bulletins on the progress, giving all available information on the subject, to the county medical societies, and then have this material published in the ILLINOIS MEDICAL JOURNAL.

J. S. Templeton, M. D.
L. E. Day, M. D.
H. P. Saunders, M. D.
H. M. Camp, M. D., Chairman.

PICTURES OF THE PAST PRESIDENTS AND SECRETARY WANTED

Officers of the Illinois State Medical Society are extremely anxious to complete the file of pictures of all past presidents. There are a number missing and it is hoped that someone may be able to furnish copies to the Editor of the JOURNAL, Charles J. Whalen, M. D., 25 East Washington Street, Chicago.

President's Name	Year
Samuel Thompson	1851
C. N. Andrews	1854
A. H. Luce	1864
J. M. Steele	1865
S. W. Noble	1867
G. W. Albin	1871
Secretary's Name	**Year**
H. Shoemaker	1851

ILLINOIS STATE MEDICAL SOCIETY COMMITTEE ON MEDICAL BENEVOLENCE

The House of Delegates of the Illinois State Medical Society at its Annual Meeting held May 21-22-23, 1940, *voted that certain changes be made in the Constitution and By-Laws to enable the Society to establish a Benevolent Fund for indigent physicians and their widows.*

The plan adopted very closely resembles the one which has been operating in Pennsylvania for the past thirty-seven years.

We are publishing herewith the personnel of the Committee together with an outline of the purposes and the power given the Committee to carry on this work.

Committee on Medical Benevolence, John S.

Nagel, Chairman 185 N. Wabash, Chicago, Ill. Charles H. Hulick, Shelbyville; Clarence H. Boswell, Rockford.

PURPOSES OF THE COMMITTEE

1. To create a Benevolence Fund:

a. Through allocation of $1.00 each year from dues of each member.

b. Through gratuities, endowments, etc.

c. Through the efforts of the Women's Auxiliary to the Illinois State Medical Society.

2. To investigate cases of alleged financial difficulties on the part of members, their widows or widowers.

3. When found worthy, to appropriate regular monthly benefits not to exceed $25.00 to $30.00 per month in any one case. When deemed advisable, may appropriate more over a short period of time when rehabilitation seems probable.

4. To designate the component society secretary in each county as the county chairman to submit applications from members for benefits, then to see that a questionnaire form is properly executed to give the desired information relative to the case. The Councilor of the District may assist the Committee in submitting names of members, their widows or widowers, when he believes the individual is entitled to the benefits herein prescribed.

5. When it is the opinion of the Committee that the case is a worthy one and benefits should be allowed, the Chairman of the Committee should notify the Secretary of the State Medical Society, stating the amount agreed upon as the regular allowance, stating the intervals at which the benefits shall be paid, so that proper vouchers may be submitted.

THE INVESTIGATIONS

When it is reported to the Committee that a member, widow or widower of a member is needy and unable to secure the necessities of life, a questionnaire form shall be submitted from the Secretary's office asking for the following information:

1. A brief social history of the applicant, past and present. Data concerning reasons for being in want whenever possible, and all other pertinent information which will enable the Committee to take the proper action.

2. A brief financial history including present assets and income, sources and amount.

3. Disbursing of present resources (rent, food, clothing, etc.).

4. Statements as to probable permanency of the present distress.

5. Any possible sources of assistance such as:

a. Relatives .....

b. Friends .....

c. Fraternal Organizations.....

d. Insurance .....

e. Pensions .....

6. Have all sources of help been solicited?

7. Additional information. Means by which influence might be exerted to find employment or some other source of income. Is there a possibility of rehabilitation? (With moderate financial assistance over a short period of time, would it be possible for the applicant to become self-supporting?)

PROCEDURE

Requests from members, their widows or widowers for assistance, if submitted to the Secretary, shall be referred to the Committee promptly. At the same time a questionnaire form will be submitted to the applicant or to the county society secretary, or to the Councilor if the information is submitted by him. All possible information which will aid the Committee in determining the eligibility for assistance, the amount actually needed, or if rehabilitation through short time payments is probable, should be submitted promptly.

Each case will receive the proper consideration by the entire committee which shall pass final judgment on:

1. Eligibility for aid.
2. The amount of aid.
3. Whether for a short time or permanently.

The decision of the Committee shall be final and there will be no higher authority within the Society to whom appeals from decisions of the Committee can be referred.

In the event that additional income is received and the individual is no longer eligible for further benefits, the county society secretary or the Councilor submitted the data, should notify the Committee of these facts promptly.

As soon as a reasonable amount is accumulated in the Benevolence Fund, only the income from the Fund shall be used to pay benefits.

The Medical Benevolence Fund shall be subject to an annual audit as are other funds of the

Illinois State Medical Society, although merely the amount of the Fund, the payments made during the year, the additions to the Fund, and the interest from investments shall be mentioned. The names of beneficiaries shall not appear in the annual audit, nor shall they be mentioned in the annual report of the Committee to the House of Delegates.

The Secretary of the State Medical Society shall maintain a separate file for all correspondence relative to beneficiaries, amounts paid, investigations and minutes of meetings of the Committee, which shall be a closed file and not open to inspection by others than members of the Committee, the Auditor, or a regularly designated Committee of the House of Delegates.

As the regular vouchers of the Illinois State Medical Society are paid through the State Bank and Trust Company of Evanston, all funds for benevolence purposes shall be maintained in another bank and payments for benevolence purposes shall constitute the only vouchers drawn on these funds. The Council of the Illinois State Medical Society has allocated th sum of $5,000.00 maintained in the National Bank of Monmouth for several years as a Certificate of Deposit, as the nucleus for the Benevolence Fund, and payments shall be made from this fund on this bank.

NOTE: The above report and procedure was presented to the Council of the Illinois State Medical Society in regular session on August 4, 1940, by the Chairman of the Committee on Medical Benevolence. The report and procedure were approved, and the Committee instructed to make the necessary arrangements to function immediately. The Council was authorized by the House of Delegates at the 1940 annual meeting to approve a method of procedure so that the work could be started with a minimum amount of delay.

AVERAGE NORMAL BLOOD PRESSURE

Age	Systolic	Diastolic	Pulse Pressure
10 years	103	70	33
15 years	113	75	38
20 years	120	80	40
25 years	122	81	41
30 years	123	82	41
35 years	124	83	41
40 years	126	84	42
45 years	128	85	43
50 years	130	86	44
55 years	132	87	45
60 years	135	89	46

A HUNDRED YEARS OLD

DO YOU KNOW—

It is not infrequently that we read in the papers about some man or woman celebrating their hundredth birthday. What profound changes have taken place in the lives of these individuals! We have the telephone, the radio, the airplane, streamliners, and hundreds of inventions which have made life simpler, or perhaps more complex.

* * *

DO YOU KNOW—

The changes in conditions relating to health have been even more pronounced than any of these centenarians could testify. In order to show some of these changes, the Illinois State Medical Society is celebrating its one hundredth birthday in Peoria this May. Exhibits will demonstrate the tremendous progress in Illinois health since 1840.

* * *

DO YOU KNOW—

A hundred years ago communicable diseases played havoc. Cholera, malaria, smallpox, yellow fever, typhoid fever, dysentery and diphtheria were common to all. The year 1839 was known as the sick year, for so much illness was prevalent along the Mississippi River that few could be induced to locate anywhere near it. The city of Peoria in 1840 had a population of 1,467. Today, with a population of more than 100,000, it is a modern, healthy city in every sense.

* * *

DO YOU KNOW—

A hundred years ago there was a high mortality among the physicians due to their exposure to contagious and infectious diseases. Today members of the Illinois State Medical Society have shown the public and have demonstrated to themselves that certain diseases can be prevented. Today we have laws protecting our milk and water supplies. Many diseases so highly prevalent in the past one hundred years are seldom seen these days.

* * *

DO YOU KNOW—

The organization of the Illinois State Medical Society and the establishment of the Illinois State Department of Public Health were two great factors in making Illinois' health record one to be envied today. No wonder the doctors of the state are proud to say they belong to an organization which has survived so many years and is able to celebrate its hundredth birthday with a record of remarkable achievement.—Educational Committee of the Illinois State Medical Society.

No need for more Government Hospitals since 1919, a total of 1,800,000 ex-service men have been admitted to Government hospitals for care. A total of 62,175 beds are now available for these men in the Government hospitals. This means one bed to every 40 former service men. It is believed that the peak load will be reached about 1949, or ten years hence.

MEDICAL ECONOMICS

H. M. Camp, M. D.
E. P. Coleman, M. D.
J. H. Hutton, M. D.
Ralph Peairs, M. D.
R. K. Packard, M. D.

Edited by the Committee on Medical Economics
of the
Illinois State Medical Society
E. S. Hamilton, M. D., Chairman
Kankakee, Illinois
Address all letters and communications to the Chairman.

C. H. Phifer, M. D.
C. B. Reed, M. D.
C. B. Ripley, M. D.
C. E. Wilkinson, M. D.
W. M. Hartman, M. D.

During the past month Washington has demonstrated no interest in reform movements, especially the Wagner bill and the so-called Hospital Bills now before Congress, particularly the Conscription Bill, and other defense measures. Under such circumstances it is not surprising that the Wagner bill and the Hospital bill have been temporarily pigeon-holed. But to the majority of the profession this respite is viewed as temporary. This is especially true if the New Deal Administration is returned to power at the November election. Meanwhile, with an election coming up and all members of the House of Representatives up for reelection as well as one third of the Senators, including one from Illinois, the medical profession is given another opportunity to get definite expression of opinion and promise as to how they will vote on any bill presented to the next Congress in regard to changing the manner in which the practice of medicine is conducted in the United States. Every medical man in the state of Illinois should make an opportunity to talk over this matter with both the Democratic Candidate, Senator Cassidy and his opponent on the Republican ticket Brooks. They will appear in practically every part of the state between now and the first Tuesday in November and will take time to discuss their views with their constituents and possibly make some definite promises, which must necessarily be evaluated by the electorate.

This Committee wishes to expreess its regret of the sudden and unexpected death of Councilor Henry G. Horstman of Murphysboro. In the year and a half, Dr. Horstman was on the Council he demonstrated an interest and knowledge of the problems of the medical profession, which was remarkable. He was always interested in the economic problems and represented the southern portion of the state in an excellent manner We hope that his successor, when selected, will continue to represent his district in the same manner and that he will give this committee the same cooperation it received from Dr. Horstman.

The writer of this column has known for some time that it has expressed his personal views and reflected his personal readings month by month. To help correct this condition, he is endeavoring to obtain articles from other members of the Committee and has tentative promises of articles from at least two members in the next two or three months. It takes time and energy to write papers, particularly each month, and the Chairman will appreciate hearing from any member of the Illinois State Medical Society, who wishes to present an article on any economic subject, remember this is your JOURNAL and the column is being conducted as a clearing house for all opinions on medical economic problems as they affect the medical profession of Illinois.

The August 17 issue of *Life* presents another article highly critical of the medical profession. We hope that many of you have read it and properly appraised it. Had it been printed in *Liberty,* one would not have been surprised, but an article so apparently exposed by critics of the medical profession and definitely dangerous to public health, is scarcely worthy of *Life.* A regular reader of *Life* must be made a little skeptical of many of the other articles which appear on its pages when one is printed as unfair as this to 180,000 potential purchasers.

Those of you who read the *Journal of the American Medical Association* are undoubtedly following the results of the Questionnaire being sent out by the AMA to all doctors of the United States in an effort to classify them according to their desires in the event of a war and also to list them according to their special qualifications. In the August 17 issue of the *J.A.M.A.,* it is reported that 80,000 physicians have returned the filled out questionnaires. Illinois

has returned 5,883 or 47.8 per cent. This is a creditable showing, but there seems to be no good reason why it should not be almost 100 per cent. The only question that may be difficult to answer is the one whether the answering physician is willing to enlist in the event of a war. It is not mandatory that an answer of yes or no be given. If for any reason the physician does not wish to answer this question he can say, "undecided at this time."

A movement is under way in some of the adjoining states to start an organization to support those in the coming presidential election, who are admittedly on record as favoring the present method of conducting the practice of medicine. This will, as we understand it be non-partisan, but will endorse and work for those friendly to the medical profession. It is difficult to evaluate any such plan until more is known about it than is available at this time, but it does seem that there is considerable to recommend some such concerted action by the medical profession at this time, when its actual existence is being threatened as it has in the past few years.

Remember that this Column is open to expressions of opinion from all members of the medical profession in the state of Illinois. Help us to make this an interesting column by writing to the Chairman and presenting your views.

E. S. Hamilton,
Chairman.

Correspondence

HISTORICAL DATA WANTED

To the Editor:

To Component County Societies of the Illinois State Medical Society: Those of you who attended the Peoria session of the Society will recall the very fine photographic exhibit arranged by Dr. Carl E. Black to set forth as fully as possible the history of the Illinois State Medical Society. A Committee on Archives is very anxious to add to this material. Further it is asking that each county society appoint a similar committee and that it secure from each of its members as much historical data as possible. This includes photographs, material from family scrap books, newspaper clippings and other material pertaining to physicians in Illinois or of those physicians who were once living in Illinois.

If you do not know what to do with your old minute books, please get them into the hands of the Committee on Archives.

Arrangements are being made to store all this material with Dr. H. M. Camp, Secretary of the Illinois State Medical Society. Your contribution should be sent to the undersigned or to Dr. Camp.

Dr. Carl E. Black, Jacksonville;
Dr. P. J. McDermott, Kewanee;
Dr. D. D. Monroe, Alton, Chairman.

FREE LABORATORY DIAGNOSTIC SERVICE

A limited laboratory service in the diagnosis of cancer is now available from the State Department of Public Health to the physicians of Illinois. Biopsy specimens taken from poor patients will be accepted and examined free at the Department's diagnostic laboratories, 1800 West Fillmore Street, Chicago. Physicians and dentists are invited to take advantage of this service in the interest of early diagnosis and subsequent treatment of patients with cancer. Specimens taken from patients who are able to pay for such service should be sent to privately operated pathological laboratories.

Physicians and dentists who decide to use this laboratory diagnostic service are urged to select the specimens for biopsy with great care. The specimen should be large enough to include the lesion and also, if possible, a small part of adjacent normal tissue. The specimen should be removed without crushing, placed immediately into 10 per cent. formalin and mailed in a tightly sealed container to the Cancer Diagnostic Service, State Department of Public Health, 1800 West Fillmore St., Chicago. Specimens should be taken, of course, only when there are reasonable grounds for suspecting tumors. Reports of examinations will be placed in the mails usually within 48 hours after receipt of specimens.

Suitable specimen containers may be obtained on request from the address given above.

At the present time the Cancer Diagnostic Service is limited practically to the laboratory examination of specimens submitted. Later, it is anticipated that the service can provide more extensive assistance, specially of an educational nature, to the medical profession in the diagnosis of cancer. Experience in tissue examina-

tion should be helpful in this respect, as time goes on. Inquiries concerning the diagnosis of cancer will be answered even now.

Dentists and oral surgeons are invited to use the diagnostic service. They often have opportunity to see in the oral cavity conditions suspicious of cancer in the early stages. Specimens might very advantageously be taken under such circumstances and submitted to the laboratory

In addition to the diagnostic service the Department has established also a Division of Cancer Control. While these two services are distinct and are administered independently, in accordance with law, they will work in close cooperation with each other and with the medical profession.

The work of the Division on Cancer Control will be educational in character. It will promote public interest in the early diagnosis and appropriate treatment of cancer and cultivate professional interest in post graduate study in the diagnosis and treatment of cancer and in the program of control.

Both of these services were established as a result of laws enacted which directed the State Department of Public Health to undertake these activities. Both laws were passed by the General Assembly in 1939 in response to popular demand.

The treatment of patients will not be undertaken by either service. The laws do not provide for the treatment of patients and appropriations are not sufficient to finance treatment facilities.

The cancer diagnostic service is a unit in the Division of Laboratories. Dr. Perry J. Melnick is the pathologist in immediate charge of the cancer diagnostic unit in the laboratory.

The Division of Cancer Control is an administrative unit of the State Department of Public Health. Dr. R. V. Brokaw is the chief of this Division. His headquarters, like that of the diagnostic service, is located at 1800 West Fillmore Street, Chicago.

WOMAN'S AUXILIARY TO THE ILLINOIS STATE MEDICAL SOCIETY

Dear Members of the Auxiliary to the Illinois State Medical Society:

As your President for the coming year, I greet you.

It is with a feeling of humility that I assume the responsibility of leadership for the ensuing year. I am fully cognizant of the splendid record made by my predecessors and I trust with the cooperation of the new President, Dr. J. S. Templeton, the Advisory Committee, Officers and Chairmen of the Board, that at the end of our stewardship, we may leave something constructive from our year's endeavor.

I am making a special appeal to the Physicians in this State of Illinois to become Auxiliary minded. Particularly the Presidents of the County Societies and the Councilors. Without their cooperation, the Auxiliary will cease to function.

If every physician had been privileged to hear the address of the President of the American Medical Association, Dr. Nathan B. Van Etten, given in New York during the convention, I feel certain that they would realize the important part their wives may be able to play. I refer to the many problems confronting the Medical Profession today.

The current and urgent problem is the menace of Socialized Medicine. This problem can be offset by discreet propaganda. To mould public opinion and to make it aware of the objectives of organized medicine must be the aim of the Auxiliary. In order to bring these objectives to fruition we must establish a program.

I urge all members to become instructed in matters pertaining to individual and public health in order that we may converse intelligently with the laity.

All members of the Auxiliary should become familiar with the platform of the American Medical Association. We were indeed edified by the address of the President of the American Medical Association, Dr. N. B. Van Etten. He said:

"I believe that the quality of your membership is such that you may be able to influence women's organizations of all kinds in a beneficial way for the service of the community and the promotion of the public health. I believe that you can matereially influence health statistics in the next decade.

"The effect of your work upon your special educational projects is sure to be felt. I am convinced that the place for this work is the small community and that the sum of such efforts will change the national picture."

I quote these few lines from Dr. Van Etten's

address because of their application and importance to our organization.

Let us in Illinois strive to make this year outstanding. Every physician's wife who is interested in her husband's profession will heed the call of the Auxiliary.

We wish all doctors' wives to become members. We hope to make our programs attractive and with your support, the Auxiliary will double in membership this year. To those who are not able to affiliate with an Auxiliary, we ask you to become members at large. Through this membership you will receive literature from the State Auxiliary which will keep you informed of the progress of the organization.

The present conditions in Europe have awakened us to the necessity of being prepared. We cherish our democracy. Let us guard it by actively mobilizing ourselves, in order to protect its principles and traditions, in which the Medical Profession has always been an integral part.

Mrs. Harry Dooley, President

Mrs. C. W. Stuart,

Chairman, Press and Publicity.

MATERNAL WELFARE COMMITTEE HAS BEEN REAPPOINTED

The Council of the Illinois State Medical Society has reappointed the Maternal Welfare Committee for the fourth consecutive year. The work of the Committee has been to create a state wide organization and to develop interest in this educational campaign on prenatal care among both physicians and the public.

The Chairman appointed a sub-committee consisting of Doctors Bitter of Quincy, Loar of Bloomington, and Lange of Belleville to revise last year's program to meet the need of modern obstetrics. This Committee completed its findings and the new program was presented to and approved by the State Committee at its July meeting. The new programs will soon be in the hands of the county chairmen for distribution to all physicians of organized medicine.

The Maternal Welfare Committee has recently been assured of the cooperation of the Ladies Auxiliary of the State Society and the Federated Woman's Clubs of Illinois and the Committee is looking forward to a most successful year.

The program for 1940 is as follows:

The Committee on Maternal Welfare of the Illinois State Medical Society feel that the adoption of the following suggestions by the County Medical Societies will lower the Maternal and Infant death rate within our State.

1. More emphasis should be placed on adequate prenatal care:

a. Monthly visits up to the seventh month, then every two weeks—history—physical examination including pelvic measurements—urinalysis—blood pressure—Kahn—blood count including red, white and hemoglobin—weight and dietary instructions.

2. We recommend that each County Medical Society appoint a Maternal and Child Welfare Committee whose duties should consist of:

a. Investigate maternal, fetal and early infant deaths for constructive study in reducing mortality. This investigation to be carried out by the County Chairmen and other physicians appointed by local Medical Society; all information pertaining to this study be kept in the hands of the Medical Profession.

b. Have an adequate number of programs on maternal welfare and pediatric subjects before local society and hospital groups to meet the need of that community.

c. Encourage the educational program among the nurses of the community by such means as moving pictures and special lectures.

d. Encourage any improvement of local hospital facilities for better maternal care.

3. We suggest that the Chairman of the Maternal Welfare Committee be designated as the County Chairman and be responsible for the furthering of this program in his respective county.

We suggest that he appoint a permanent Maternal Welfare Committee composed of professional and lay groups to further the program of lay education.

4. Encourage post-graduate work and refresher courses among the physicians and promote educational facilities in Hospital, County and State in Obstetrics and Pediatrics.

5. Encourage consultation in all Obstetrical complications.

T. B. Williamson, M. D., Chairman.

John F. Carey, M. D., Secretary.

PLEASE IDENTIFY PHOTOGRAPHS

Jacksonville, Illinois.
July 28, 1940.

To The Editor:

Many photographs of Illinois physicians were handed in at the annual meeting of the Illinois State Medical Society in May and others have been sent to me here. In a number of cases there was insufficient data to show who sent them.

I am publishing this notice in the ILLINOIS MEDICAL JOURNAL notifying those who have not received due acknowledgement to write me and I will be glad to express appreciation.

There is one set of six or eight excellent photographs that I do not know who handed them in. I would like to show these people proper appreciation. Publishing this item in the ILLINOIS MEDICAL JOURNAL will at least show the right spirit.

Carl E. Black, M. D.

FALL MEETING OF IOWA AND ILLINOIS

The fall meeting of the Iowa and Illinois Central District Medical Association will be held Wednesday evening, September 18, at the Le Claire Hotel in Moline, Illinois.

Preceding the scientific meeting a motion picture in color of the picnic, which was held June 5, will be shown.

The scientific program will begin at 8 P. M. with a ten-minute paper by Dr. Earl B. Ritchie of Davenport, "A Short Resume on Industrial Dermatosis."

The principal address of the evening will be given by Dr. Percy S. Pelouze of Philadelphia, Pa., who will speak on "Present Day Views on Gonorrhea and Its Treatment."

A dinner will precede the meeting at 6 P. M.

LABORATORIES NEWLY APPROVED FOR PRE-MARITAL TESTS

Alexian Brothers Hospital Laboratory, 1200 Belden Ave., Chicago. Formerly approved for Kahn only.

P. Caccia Laboratory, 528 S. Halsted St., Chicago.

Evangelical Deaconess Hospital Laboratory, 7th & Walnut, Lincoln.

C. H. Hulick, M.D. & D. Biddlecomb, M.D. Laboratory, 2003 Broadway, Shelbyville (Gc only).

Dr. Philip H. Kreuscher & Dr. Richard J. Bennett, Jr. Lab., 208 S LaSalle St., Chicago.

Modern Medical Laboratory, Branch No. 1, 9204 Commercial Ave., Chicago.

St. Charles' Hospital Lab., New York & Fourth Sts., Aurora. Formerly approved for Kahn only.

St. Francis Hosiptal Lab., 513 Elliott St., Kewanee (Gc only).

St. Joseph Hospital Lab., Jefferson St., Elgin.

LABORATORIES NEWLY APPROVED FOR PNEUMONIA TYPING

C. H. Hulick, M.D. & D. Biddlecombe, M.D. Laboratory, 2003 Broadway, Shelbyville.

Mark Greer Hospital Lab., Vandalia.

People's Hospital Lab., 925 West Avenue, Peru.

INTERNATIONAL MEDICAL ASSEMBLY

Inter-State Postgraduate Medical Association of North America

October 14-18, 1940.

Pre-Assembly clinics, Saturday, October 12; Post-assembly clinics, Saturday, October 19—Cleveland Hospitals.

CLEVELAND, OHIO

Monday, October 14

8:00 A.M.

Diagnostic Clinic: "Surgical Management of Recurrent Hyperthyroidism"—Dr. Richard B. Cattell, Lahey Clinic, Boston, Massachusetts.

Diagnostic Clinic: "Types and Treatment of Chronic Nephritis"—Dr. John Musser, Professor of Medicine, Tulane University School of Medicine, New Orleans, Louisiana.

Diagnostic Clinic: "Treatment of Bronchiectasis"—Dr. John Alexander, Professor of Surgery, University of Michigan School of Medicine, Ann Arbor, Mich.

Intermission for Review of Exhibits

Diagnostic Clinic: "Adrenal Insufficiency and the Use of Synthetic Adrenal Cortical Hormone"—Dr. George W. Thorn, Associate Professor of Medicine, Johns Hopkins University School of Medicine, Baltimore, Md.

Diagnostic Clinic: "Prevention of Deformities in Arthritis"—Dr. Loring T. Swaim, Boston, Mass.

Noon Intermission

1:00 P.M.

Diagnostic Clinic: "Surgical Treatment of Peptic Ulcer"—Dr. William F. Rienhoff, Associate Professor of Surgery, Johns Hopkins University School of Medicine, Baltimore, Md.

Diagnostic Clinic: "Clinical Types of Pituitary Disease"—Dr. David P. Barr, Busch Professor of Medicine, Washington University School of Medicine, St. Louis, Mo.

Address: "Postoperative Management of the Surgical Patient"—Dr. Frederick A. Coller, Professor of Surgery, University of Michigan School of Medicine, Ann Arbor, Mich.

Address: "Some Observations on the Nature of Acute Nephritis"—Dr. John P. Peters, John Slade Ely,

Professor of Medicine, Yale University School of Medicine, New Haven, Conn.

Intermission for Review of Exhibits

Address: "The Hazards of Pregnancy and Labor in the Grande Multipara"—Dr. Nicholson J. Eastman, Professor of Obstetrics, Johns Hopkins University School of Medicine, Baltimore, Md.

Address: "Recent Advances in Chemotherapy"—Dr. Chester S. Keefer, Associate Professor of Medicine, Harvard Medical School; Wade Professor of Medicine, Boston University School of Medicine, Boston, Mass.

Address: "Treatment of Wounds"—Dr. Frederick Christopher, Associate Professor of Surgery, Northwestern University School of Medicine, Evanston, Ill.

Address: "Intestinal Absorption As a Clinical Physiological Problem"—Dr. Maurice B. Visscher, Professor of Physiology and Head of the Department, University of Minnesota Medical School, Minneapolis, Minn.

Dinner Intermission

7:00 P.M.

Address: "The Interpretation and Treatment of Spells of Unconsciousness in Medical and Surgical Practice"—Dr. Soma Weiss, Hersey Professor of the Theory and Practice of Physic, Harvard Medical School, Boston, Mass.

Address: "Encephalopathies in Children"—Dr. Bronson Crothers, Assistant Professor of Pediatrics, Harvard University Medical School, Boston, Mass.

Address: "Surgery of Hypoglycemia with Special Reference to Resection of the Pancreas"—Dr. Vernon C. David, Clinical Professor of Surgery, Rush Medical College, Chicago, Ill.

Address: "The Immediate Treatment of Head Injuries"—Dr. Donald Munro, Assistant Professor of Neurological Surgery, Harvard University Medical School, Boston, Mass.

Address: "Management of Pelvic Inflammatory Disease"—Dr. John R. Fraser, Professor of Obstetrics and Gynecology, McGill University Faculty of Medicine, Montreal, Canada.

Address: "Common Errors in Cardiac Diagnosis"—Dr. A. Carlton Ernstene, Head of the Cardiorespiratory Department, Cleveland Clinic, Cleveland, Ohio.

Tuesday, October 15

8:00 A.M.

Diagnostic Clinic: "The Use of Testosterone Propionate in Male Hypogonadism"—Dr. E. Perry McCullagh, Cleveland Clinic, Cleveland, Ohio.

Diagnostic Clinic: "Special Problems in the Nutrition of Children"—Dr. Henry J. Gerstenberger, Professor of Pediatrics, Western Reserve University School of Medicine, Cleveland, Ohio.

Diagnostic Clinic: "Immediate Treatment of Burns"—Dr. Donald M. Glover, Associate Clinical Professor of Surgery, Western Reserve University School of Medicine, Cleveland, Ohio.

Intermission for Review of Exhibits

Diagnostic Clinic: "Medical Treatment of Peptic-Ulcer"—Dr. Walter C. Alvarez, Professor of Medicine, University of Minnesota Graduate School of Medicine, Mayo Clinic, Rochester, Minn.

Diagnostic Clinic: "Fractures of the Neck of the Femur"—Dr. William R. Cubbins, Clinical Professor of Bone and Joint Surgery, Loyola University School of Medicine, Chicago, Ill.

Noon Intermission

1:00 P.M.

Diagnostic Clinic: "Gout"—Dr. Russell L. Haden, Cleveland Clinic, Cleveland, Ohio.

Diagnostic Clinic: "Surgery of the Pancreas"—Dr. Waltman Walters, Professor of Surgery, University of Minnesota Graduate School of Medicine, Mayo Clinic, Rochester, Minn.

Address: "Medicine and Surgical Aspects of the Obstructing Prostate"—Dr. Hugh H. Young, Professor of Urology, Johns Hopkins University School of Medicine, Baltimore, Maryland.

Address: "Protruded Intervertebral Disc"—Dr. Howard C. Naffziger, Professor of Surgery, University of California School of Medicine, San Francisco, Calif.

Intermission for Review of Exhibits

Address: "Allergic and Non-Allergic Treatment of Asthma"—Dr. Warren T. Vaughan, Vaughan-Graham Clinic, Richmond, Va.

Address: "Tumors of the Kidney in Children"—Dr. Herman L. Kretschmer, Clinical Prof. of Surgery, (Genito-Urinary), Rush Medical College, Chicago, Ill.

Address: "Cancer of the Stomach"—Dr. Howard K. Gray, Assistant Professor of Surgery, University of Minnesota Graduate School of Medicine, Mayo Clinic, Rochester, Minn.

Address: "Treatment and Complications of Whooping Cough"—Dr. Gerald S. Shibley, Associate Clinical Prof. of Medicine, Western Reserve University School of Medicine, Cleveland, Ohio.

Dinner Intermission

7:00 P.M.

Address: "The Treatment of Vesico-Colonic Fistula"—Dr. Charles W. Mayo, Assistant Professor of Surgery, University of Minnesota Graduate School of Medicine, Mayo Clinic, Rochester, Minn.

Address: "General Problems of Old Age"—Dr. Lewellys F. Barker, Professor Emeritus of Medicine, Johns Hopkins University School of Medicine, Baltimore, Md.

Address: "Coronary Artery Disease"—Dr. Roy W. Scott, Professor of Clinical Medicine, Western Reserve University Medical School, Cleveland, Ohio.

Address: "Modern Treatment of Scoliosis"—Dr. Alan deForest Smith, Clinical Prof. of Orthopedic Surgery, Columbia University College of Physicians and Surgeons, New York, N. Y.

Address: "The Treatment of Acute Traumatic Intracranial Hemorrhage"—Dr. Eric Oldberg, Professor and Head of the Department of Neurology and Neurological Surgery, University of Illinois College of Medicine, Chicago, Ill.

Address and Movie: "Successful Defibrillation of the Human Ventricles, the Establishment of a Resuscitation

Squad in our Hospitals"—Dr. Claude S. Beck, Associate Prof. of Surgery, Western Reserve University School of Medicine, Cleveland, Ohio.

Wednesday, October 16

8:00 A.M.

Diagnostic Clinic: "Pre-Symptom Tuberculosis"—Dr. Raymond C. McKay, Assistant Clinical Prof. of Medicine, Western Reserve University School of Medicine, Cleveland, Ohio.

Diagnostc Clinic: "Cancer of the Rectum"—Dr. Thomas E. Jones, Cleveland Clinic, Cleveland, Ohio.

Diagnostic Clinic: "The Various Forms of Edema with the Mechanism of Production and Treatment"—Dr. Italo F. Volini, Clinical Professor of Medicine, Loyola University School of Medicine, Chicago, Ill.

Intermission for Review of Exhibits

Diagnostic Clinic: "Hernia"—Dr. W. Wayne Babcock, Professor of Surgery and Clinical Surgery, Temple University School of Medicine, Philadelphia, Pa.

Diagnostic Clinic: "Diagnosis and Management of Hepatocellular Jaundice"—Dr. Henry L. Bockus, Professor of Gastro-Enterology, University of Pennsylvania Graduate School of Medicine, Philadelphia, Pa.

Noon Intermission

1:00 P.M.

Diagnostic Clinic: "Contact Dermatitis and Its Differentiation"—Dr. Frank C. Knowles, Professor of Dermatology, Jefferson Medical College, Philadelphia, Pa.

Diagnostic Clinic: "Deficiency Syndromes Commonly Seen in America"—Dr. James S. McLester, Professor of Medicine, University of Alabama School of Medicine, Birmingham, Ala.

Address: "Surgery of the Gallbladder and the Bile Ducts"—Dr. Roscoe R. Graham, Assistant Professor of Surgery, University of Toronto Faculty of Medicine, Toronto, Canada.

Address: "Esophagology in Relation to General Medicine"—Dr. Chevalier Jackson, Honorary Professor of Broncho-Esophagology, Temple University School of Medicine, Philadelphia, Pa., and Dr. Chevalier L. Jackson, Professor of Broncho Esophagology, Temple University School of Medicine, Philadelphia, Pa.

Intermission for Review of Exhibits

Address: "Common Errors in the Selection of Patients for Surgery"—Dr. Irvin Abell, Clinical Professor of Surgery, University of Louisville School of Medicine, Louisville, Ky.

Address: "Poliomyelitis, Early Diagnosis and Treatment"—Dr. John A. Toomey, Associate Professor of Pediatrics, Western Reserve University School of Medicine, Cleveland, Ohio.

Address: "Endocrine Factors in Gynecological Disease"—Dr. Otto H. Schwarz, Professor of Clinical Obstetrics and Gynecology, Washington University School of Medicine, St. Louis, Mo.

Address: "The Management of the Acute Abdomen in Children"—Dr. Charles H. Phifer, Professor of Surgery, University of Illinois School of Medicine, Chicago, Ill.

7:00 P.M.

ASSEMBLY DINNER

For members of the profession, their ladies and friends.

Informal

Dr. Chevalier Jackson, President of the Inter-State Postgraduate Medical Association of North America —Master of Ceremonies.

Addresses by:

Dr. Ross T. McIntire, Surgeon-General, United States Navy, Washington, D. C.

Dr. Nathan B. Van Etten, President, American Medical Association, New York, N. Y.

Other distinguished citizens of the world.

Thursday, October 17

8:00 A.M.

Diagnostic Clinic: "Removal of Right Cerebral Hemisphere for Infiltrating Glioma"—Dr. Louis Karnosh, Associate Clinical Professor of Nervous Diseases, Western Reserve University School of Medicine, Cleveland, Ohio, and Dr. W. James Gardner, Chief of Neurosurgery, Cleveland Clinic, Cleveland, Ohio.

Diagnostic Clinic: "Medical and Surgical Aspects of Chronic Ulcerative Colitis"—Dr. Henry Cave, Assistant Clinical Professor of Surgery, Columbia University College of Physicians and Surgeons, New York, N. Y., and Dr. Thomas Mackie, Assistant Clinical Professor of Medicine, Columbia University College of Physicians and Surgeons, New York, N. Y.

Diagnostic Clinic: "Obesity"—Dr. Robert W. Keeton, Professor of Medicine, University of Illinois College of Medicine, Chicago, Ill.

Intermission for Review of Exhibits

Diagnostic Clinic: "Diverticulitis"—Dr. Claude F. Dixon, Associate Professor of Surgery, University of Minnesota Graduate School of Medicine, Mayo Clinic, Rochester, Minn.

Diagnostic Clinic: "Gold Therapy in Rheumatoid Arthritis"—Dr. Russell L. Cecil, Professor of Clinical Medicine, Cornell University Medical College, New York, N. Y.

Noon Intermission

1:00 P.M.

Diagnostic Clinic: "Sugical Treatment of Peripheral Vascular Disease"—Dr. Alfred W. Adson, Professor of Neurosurgery, University of Minnesota Graduate School of Medicine, Mayo Clinic, Rochester, Minn.

Diagnostic Clinic: "Diagnosis and Treatment of Myxedema"—Dr. Cyrus C. Sturgis, Professor of Internal Medicine, University of Michigan School of Medicine, Ann Arbor, Mich.

Address: "Acute Surgical Abdomen"—Dr. Elliott C. Cutler, Moseley Professor of Surgery, Harvard University Medical School, Boston, Mass.

Address: "Infections of the Urinary Tract"—Dr.

William F. Braasch, Professor of Urology, University of Minnesota Graduate School of Medicine, Mayo Clinic, Rochester, Minn.

Intermission for Review of Exhibits

Address: "Malignancy of the Colon"—Dr. Charles Gordon Heyd, Clinical Professor of Surgery, New York Postgraduate Medical School, New York, N. Y.

Address: "Relationship of Ophthalmology to Systemic Disease"—Dr. William L. Benedict, Professor of Ophthalmology, University of Minnesota Graduate School of Medicine, Mayo Clinic, Rochester, Minn.

Address: "Clinical Report and Evaluation of Low Temperature in Treatment of Cancer"—Dr. Temple Fay, Professor of Neurology and Neurosurgery, Temple University School of Medicine, Philadelphia, Pa.

Address: "Choice of Anesthesia"—Dr. John S. Lundy, Professor of Anesthesia, University of Minnesota Graduate School of Medicine, Mayo Clinic, Rochester, Minn.

Dinner Intermission

7:00 P.M.

Address: "Treatment of the Menopause"—Dr. Elmer Sevringhaus, Professor of Medicine, University of Wisconsin School of Medicine, Madison, Wis.

Address: "Physiological and Clinical Aspects of Intubation of the Small Intestine"—Dr. W. Osler Abbott, Philadelphia, Pennsylvania.

Address: "Surgical Aspects of Gastro-Intestinal Hemorrhage"—Dr. Eldridge L. Eliason, John Rhea Barton, Professor of Surgery, University of Pennsylvania School of Medicine, Philadelphia, Pa., and Dr. Julian Johnson, Philadelphia, Pa.

Address: "Treatment of Luetic Aortitis"—Dr. James E. Paullin, Professor of Clinical Medicine, Emory University School of Medicine, Atlanta, Ga.

Address: "Benign Lesions of the Neck" (slides)—Dr. Robert S. Dinsmore, Cleveland Clinic, Cleveland, Ohio.

Friday, October 18

8:00 A.M.

Diagnostic Clinic: "Leukemia, Diagnosis, and Treatment"—Dr. Claude E. Forkner, Assistant Professor of Clinical Medicine, Cornell University School of Medicine, New York, N. Y.

Diagnostic Clinic: "Shoulder Joint Injuries"—Dr. John J. Moorhead, Professor of Clinical Surgery, New York Postgraduate Medical School, New York, N. Y.

Diagnostic Clinic: "Diseases of the Aorta"—Dr. Wallace M. Yater, Professor of Medicine and Director of the Department, Georgetown University School of Medicine, Washington, D. C.

Intermission for Review of Exhibits

Diagnostic Clinic: "Diabetes: Its Complications"—Dr. Elliott P. Joslin, Clinical Professor of Medicine, Harvard University Medical School, Boston, Mass.

Diagnostic Clinic: "Surgery of the Esophagus"—Dr. Frank H. Lahey, Lahey Clinic, Boston, Mass.

Noon Intermission

1:00 P.M.

Diagnostic Clinic: "Special Problems in Surgery of the Aged Patient"—Dr. George Crile, Sr. and Dr. George Crile, Jr., Cleveland Clinic, Cleveland, Ohio.

Diagnostic Clinic: "Tumors of the Breast"—Dr. John F. Erdmann, Attending Surgeon, New York Postgraduate Medical School, New York, N. Y.

Diagnostic Clinic: "Treatment of Pernicious Anemia"—Dr. George R. Minot, Professor of Medicine, Harvard University Medical School, Boston, Mass.

Intermission for Review of Exhibits

Address: "The Modern Treatment of Congestive Heart Failure"—Dr. George Herrmann, Professor of Clinical Medicine, University of Texas School of Medicine, Galveston, Texas.

Address: "Pathological Lesions of the Larynx" (colored Movie)—Dr. Dean M. Lierle, Professor and Head of Otolaryngology, State University of Iowa College of Medicine, Iowa City, Iowa.

Address: "Herniated Nucleus Pulposis; Its Symptoms and Diagnosis"—Dr. Bernard H. Nichols, Cleveland Clinic, Cleveland, Ohio.

Tuberculosis in the Aged—It is known that when old people are found to have tuberculosis it is almost impossible to teach them to take care of themselves and protect others from infection. They will not cover their mouths during a cough or sneeze, nor will they try to protect or destroy their sputum. Their idea is that they have lived all these years with this old cough, it will not hurt them and they do not see how they can hurt anyone else. Elderly people with a chronic cough and positive sputum are a menace to society and should be isolated. C. L. Harrell, M.D., Virginia Med. Monthly, November, 1939.

The early symptoms of renal tuberculosis are not appreciated, and genito-urinary tuberculosis is regarded as a disease by itself and not as a manifestation of a generalized tuberculous condition. As a result, adequate convalescence and expert after-care are not insisted upon. Sanatorium treatment and continued supervision after operation or local treatment will favorably influence the general prognosis. J. Carver, M.D., Tubercle, April, 1939.

Chronic non-specific pulmonary disease is complex and confusing both clinically and anatomically. An increase in the density of the pulmonary markings, ring shadows, the displacement of organs or chronic pneumonia should arouse the suspicion of bronchiectasis. Paul Andrus, M.D., Amer. Rev. of Tuber., January, 1940.

Sulfanilimide and Tubercle Bacilli—The "wonder drug"—sulfanilimide—is being modified in the attempt to make it effective against the acid-fast tubercle and leprosy bacilli, but the work does not, as yet, permit any conclusion as to the efficacy of the new product in man. It is a combination of sulfanilimide and of cocoanut oil which it is hoped will enable the drug to penetrate the waxy content of the bacilli, which has heretofore served as armor against chemotherapy. Crossley, M. L., with Northey, E. H., and Hultquist, M. E., N. Y. Times, April 7, 1939.

Original Articles

ELECTROSURGICAL OBLITERATION OF THE GALLBLADDER WITHOUT DRAINAGE

(A Report of the Results of 980 Cases with a Mortality of 0.3 Per Cent.)

MAX THOREK, M. D., F. I. C. S.

Prof. of Surgery, Cook County Graduate School of Medicine; Attending Surgeon, Cook County Hospital; Surgeon-in-Chief, American Hospital

CHICAGO

Since the object of every surgical procedure is to reduce mortality and morbidity, it will, perhaps, be well to first survey the mortality rate following classical operations on the gallbladder.

Mortality Following Gallbladder Operations: In 1923, Enderlen and Hotz[1] collected 12,144 cases from the leading contemporary surgical clinic for the purpose of ascertaining the average mortality following classical cholecystectomy. The conclusion these observers have drawn from this statistical study was that in this series of 12,144 cases the mortality was 9.6 per cent. They termed this "global mortality."

In 1930 R. H. Miller[2] reported 200 consecutive cases operated on for acute cholecystitis in which stones were present in 160 cases. There were 27 deaths or 13.5 per cent. In 14 of these, cholecystectomies were done and in 13, cholecystostomies. In the same year, R. L. Sanders[3] analyzed the end-results of 500 cases of cholecystectomy with a mortality of 4 per cent. In cases which he drained the mortality was 6 per cent. Federov[4] reports a mortality of 10 per cent. in 515 cases, and in the last 228 operations, 5 per cent. In 1931, Judd and Priestley[5] in 579 cases of cholecystectomy for chronic Cholecystitis, the mortality was 1.7 per cent. N. Lindberg reports 566 patients with acute cholecystitis in which the mortality for cholecystectomy was 6.8 per cent. In 20 patients admitted in poor condition the mortality was 50 per cent. and in 9 cases of acute perforation it was 55 per cent.

From the services of Cook County Graduate School of Medicine, Cook County Hospital and American Hospital, Chicago, Illinois.

Waltman Walters[7] wrote convincingly on the newer concepts in the management of acute cholecystitis.

H. Michaelson[8] reported 712 cases between 1913 and 1935; of these 422 were cholecystectomies with a mortality rate of 3.3 per cent.; in 258 cholecystectomies drainage of the common and hepatic ducts were done in 11 cases; in 20 cholecystectomies there was a mortality of 30 per cent. In the entire group of 712 patients operated upon 7.1 per cent. died immediately after the operation; 7 after an interval operation and 44 after operation during the acute attack. Cattell[9] in 1929 noted a mortality of 6.8 per cent. in 311 cases following cholecystectomy and in 39 cases of cholecystostomy; in the last two years, the rate was less than 1 per cent. Von Haberer[10] operated on 58 patients between 60 and 70 years of age in which protracted drainage of the biliary passages was resorted to with a mortality of 27 per cent. In old patients operated on in the interval between attacks, the mortality was 8 per cent. Carter, Heyd and Holtz[11] in a study of 3,986 cases of various operative procedures give an average mortality for all biliary tract surgery of 7.7 mortality, following cholecystostomy with that of cholecystectomy. Thus, Barney Brooks and Thomas E. Wyatt[12] observed 512 operations at the Vanderbilt University Hospital for 12 years and recorded the following results:

> Primary cholecystectomy for disease limited to the gallbladded; 2.6 per cent. Primary cholecystostomy for disease limited to the gallbladder: 14.8 per cent.

Eldridge L. Eliason,[13] in a series of 537 cases in 12 years, found the mortality was 8.0 per cent. while Charles G. Heyd[14] reported in 500 noncomplicated cholecystectomies a mortality of 3.3 per cent.; again in 34 cholecystostomies, the mortality was 14.2 per cent. Heyd points out that private patients fare better than clinic cases: thus in 417 private patients there was a mortality of 4.8 per cent. while in 140 clinical cases, the mortality was 13.5 per cent. Ralph Colp and Leon Ginzburg's paper[15] was discussed by Graham of Brooklyn who pointed to a 6.6 per cent. mortality in a series of 135 cases, while Frank Lahey[16] in a large series of cases (2,346), 32 of which were complicated by stones in the common duct, the mortality in the former was 3.8

per cent. and in the latter 9.0 per cent. Richland Holtz's series[17] shows the following:

Cholecystectomy	428	517 cases	7.47	per cent.	8.82
with Choledochostomy	89		14.60	per cent.	
Cholecystostomy	45		28.80	per cent.	29.63
with Choledochostomy	9	57 cases	33.33	per cent.	

Already in 1924, Blalock[18] in a statistical study of 880 cases of biliary tract disease treated in the surgical division of the Johns Hopkins Hospital (from 1889 to 1921) considered that this series seemed almost ideal for comparison between cholecystectomy and cholecystostomy, inasmuch as 49 per cent. of the patients that were followed after leaving the hospital had the gallbladder removed and in 51 per cent. cholecystostomy was done. The percentage of cured and improved patients following the two types of operations were about the same but only 39 per cent. of the deaths followed cholecystectomy, whereas 61 per cent. of the deaths followed drainage of the gallbladder only. In addition, 79 per cent. who had recurrence of the symptoms (with and without another operation) had had the gallbladder drained at the original operation. Cholecystostomy had first been performed on 89 per cent. of the patients who had to undergo a second operation. The facts presented by this study led Blalock to believe that the gallbladder should be removed in all cases in which it is diseased except in cases with malignant disease of the biliary tract. Finally, mortality following cholecystectomy is affected by numerous extrinsic factors, and while recent reports from the best clinics show an immediate mortality of only 1 to 2 per cent., the global mortality of Enderlen and Hotz (12,144 cases) ranges around 9.6 per cent. also Verbrycke[19] states that in the Garfield Memorial Hospital, Washington, D. C., the mortality rate for biliary tract surgery, by individual operators there, during a period of two years ranged from 1 to 28 per cent.! Mortality rapidly advances with age; thus, in the sixth and seventh decades the mortality rate is more than double that of the fourth.

Wilson, Lehman and Goodwin[20] report 610 consecutive cases of cholecystectomy and cholecystostomy between 1921 and 1935 with 22 hospital deaths or a mortality of 3.6 per cent. Branch and Zollinger[21] in a series of 235 cases of cholecystitis in the Peter Bent Brigham Hospital, Boston, show the following mortality:

Total operative mortality	10.7 per cent.
Immediate operation	20.5 per cent.
Delayed operation	8.7 per cent.

G. J. Heuer[22] urges early operation for nonmalignant cases. In 36,623 cases operated on by 21 surgeons the mortality ranged between 2.6 per cent. to 10.4 per cent. or an average of 6.6 per cent. In 1,274 cases of acute inflammation of the gallbladder cholecystectomy was done by 8 surgeons with a mortality ranging between 4.7 per cent. to 22.5 per cent.—and an average of 8.7 per cent. Mackey[23] in 243 cases of cholecystectomy observed between 1922 and 1931, a mortality of 3 per cent. was recorded. There were 243 cases of cholecystitis without stones and in 21 cases cholesterosis without stone; while Eddington in 200 cases of biliary tract surgery records a mortality of 12 per cent. Bergh's critical review of 2,804 cases between 1929 and 1937, of acute cholecystitis[24] is very illuminating.

Mortality of 532 surgically treated cases of nonmalignant biliary tract disease at University of Minnesota Hospitals:

Procedure	Cases	Deaths	Mortality
Cholecystectomy (chronic and subsided cases)	435	8	1.8 per cent.
Emergency (chronic and subsided)	435	8	1.8 per cent.
Cholecystostomy (chronic cholecystitis)	10	1	10.0 per cent.
Removal of common duct stones	73	9	12.1 per cent.
Repair of stricture of common duct (Cases with asociated jaundice)	9	2	22.2 per cent.
Total	532	22	4.1 per cent.

Mirizzi[25] had no mortality in 100 cases done under the control of cholangiography while Cheever[26] in 260 consecutive cholecystectomies excluding operation on the common duct, the mortality was 0.8 per cent. Pennoyer[27] reports from the Roosevelt Hospital, New York City, 300 consecutive cases with 30 deaths or 10 per cent. (policy of hospital to wait until acute symptoms subside) while Estes[28] speaks of a 2.8 per cent. mortality in "partial" cholecystectomy as compared with 3.2-13.5 per cent. deaths resulting from classical cholecystectomy in acute cases.

MORTALITY FOLLOWING EARLY OPERATION FOR ACUTE CHOLECYSTITIS

Author	Cases	Deaths	Mortality Per Cent.	Operated On
Mentzer	21	8	39.0	within 24 hrs.
Mentzer	21	2	9.5	after 24 hrs.
Graham, H. F.	20	1	5.0	within 48 hrs.
Graham, H. F.	178	11	6.2	after 48 hrs.
Zinninger	12	0	0	within 48 hrs.
Zinninger	23	3	13.0	after 48 hrs.
Taylor	19	1	5.2	within 48 hrs.
Taylor	83	16	19.2	after 48 hrs.
McKenty	14	0	0	within 48 hrs.
Heuer	153	7	3.2	within 48 hrs.
Heuer	284	30	10.5	after 48 hrs.
Zinninger	15	1	6.6	from 3rd to 5th day
Zinninger	8	2	25.0	after 5th day
Taylor	20	1	5.0	from 3rd to 5th day
Taylor	65	15	23.8	after 5th day

Five hundred and two cases of perforation of the gallbladder—20 surgeons—15 per cent. to 65 per cent.—average 46 per cent.

Two hundred cases of acute cholecystitis operated on during the last 16 months—New York hospitals—three deaths of pulmonary complications, one cardiac failure, and one acute pancreatitis. Eliason and Ferguson[29] found in a total of 100 cases eight operative deaths—8 per cent.; while DeCourcy[30] found in 336 consecutive cholecystectomies (300 were drained) 20 deaths or 6.16 per cent. and in 36 (not drained) one death or 2.7 per cent. Bryan[31] observed in 233 cholecystectomies (Vanderbilt School of Medicine) nine deaths (four fatal cases were operated upon during an acute attack); while Doran, Lewis, Denneen and Hanssen[32] observed in Bellevue Hospital, Fourth Surgical Division (1927-1932), 200 cases on wards with 7 per cent. mortality (post-operative pneumonia, five cases; shock, three; pulmonary embolus, three; cardiac decompensation one; postoperative hemorrhage, one; massive collapse of lung, one), Shearer[33] (Barnes Hospital) notes in all biliary surgery the mortality of cholecystectomy was reduced from 6 per cent. to 4 per cent. "by treating preoperatively with glucose and calcium the patient with excessive dye retention" (Graham's test). Again Noble[34] in 194 cases had no mortality. Boyce, Veal and McFetridge[35] point out that brilliant individual surgeons and exceptionally well organized clinics report mortality rates of 1 to 3 per cent. In the Charity Hospital in New Orleans for a three year period ending December 31, 1934, in 404 cases of gallbladder disease there were 37 deaths or 9.1 per cent. (40 different surgeons), 242 cases (6 surgeons), 13.3 per cent.; 162 cases (34 surgeons), 7.4 per cent. In the first group are included all cases of empyema, rupture and gangrene of the gallbladder.

Analysis of 100 Deaths—Percentage

	Cases	Deaths	Rates
Cholecystectomy	331	20	6.0
Cholecystostomy	50	9	18.0
Choledochostomy	20	6	30.0
Other procedures	3	2	66.6
Drainage	66	11	16.6
Appendectomy also	129	8	6.2

Geissendorfer[36] reports 50 cholecystectomies performed from 1924 to 1934 on persons ranging from 20 to 70 years of age with a mortality of 28 per cent. J. M. Black[37] reported 100 cases of gallbladder operations as follows:

	Cases	Deaths	Per cent.
Acute cholecystitis	14	3	21
Chronic cholecystitis (with stones)	50	6	12
Chronic cholecystitis (without stones)	34	0	0
Carcinoma of gallbladder	2	1	50
Total	100	10	10

Operations:	Cases	Deaths
Cholecystectomy with drainage of common duct	3	1
Cholecystectomy without drainage of common duct	56	4
Cholecystostomy	25	1
Total	84	6

Salzmann and Meranze[38] report operations on 133 patients (Mt. Sinai Hospital) 1930-1935 as follows:

118 patients—gallbladder disease cholecystitis with stone
100 cholecystectomy—6 deaths
8 cholecystostomy—3 deaths
88 patients with subchronic or chronic cholecystitis with stone cholecystectomy—6 deaths—6.8 per cent.

Welcher[39] (Pels Leusden Clinic) reports:

Total gallbladder cases—1,103 (not operated on—258).
Cholecystectomy:
Lithiasis .....503— 8.15 per cent.
Empyema ..... 130—16.62 per cent.
Cholecystitis ..... 72— 5.56 per cent.
Perforation ..... 11—27.27 per cent.
Total ..... 716—14.04 per cent.

Cholecystectomy in cholelithiasis in last 3 years—1.2 per cent.
Early operation .....255; deaths—18—7.06 per cent.
Late operation .....226; deaths—16—7.08 per cent.
Interval operation .....156; deaths—10—6.41 per cent.
Attack and after.....258; deaths—19—7.3 per cent.
Age, 11-20; No. of cases, 10; deaths, 0—0 per cent.
Age, 21-30; No. of cases, 85; deaths, 4—4.75 per cent.
Age, 31-40; No. of cases, 133; deaths, 5—3.75 per cent.
Age, 41-50; No. of cases, 138; deaths, 18—12 per cent.
Age, 51-60; No. of cases, 100; deaths, 13—12 per cent.
Age, 61-70; No. of cases, 32; deaths, 6—15 per cent.

Empyema Cases—Ages as Above

1; deaths, 0— 0 per cent.
13; deaths, 2—15 per cent.
40; deaths, 5—12.5 per cent.
29; deaths, 3—10.4 per cent.
35; deaths, 6—17.1 per cent.
12; deaths, 3—25.0 per cent.

112 cholecystectomies in empyema with stone—10 deaths—8.9 per cent. (last 5 years—5 per cent). 18 cholecystectomies in empyema without stone—8 deaths—42.2 per cent. 24 cases of perforation—10 deaths—41.5 per cent.

Cannaday reports[40] 674 gallbladder cases as follows:

	Deaths	Mortality	PerCent.
Cholecystectomies	519	28	5.37
Cholecystostomies	135	8	5.92
Exploration of common duct	15	2	13.33
Cholecystogastrostomies	5	0	0

While Lamson (Seattle, Washington),[41] in 152 cases had five fatalities, Goldish and Gillespie[42] in 37 operations on the gallbladder reported 40

deaths or 5.9 per cent. They reviewed the literature up to 1933 and recorded as follows:

Author	Cases	Deaths	Per Cent.
Cave	470	32	15.3
Clark	108	5	4.6
Cahill	634	35	5.5
Dahl-Iverson	146	..	..
Danzig	113	5	4.3
Davis	156	4	2.7
De Conrey	64	4	6.2
Eliason and Ferguson	136	10	7.4
Fallon	800	37	4.6
Fowler	422	..	..
Hitzrot and Cornell	400	37	9.6
Hueck	263	19	14.0
Johnson and Pearce	192	12	6.3
Judd and Parker	989	15	1.5
Verbrycke	302	21	6.9
Sanders	500	20	4.0
Goldish and Gillespie	670	40	5.9

It has been pointed out that in competent hands the removal of the gallbladder by the time honored classical scalpel method where no complications exist, and in individuals under the age of 40, the mortality is about 2 per cent. As seen above, from some clinics even lower mortality figures are reported. It is different, it will be admitted, in individuals past 40, in the presence of complications and in the hands of the inexpert or occasional operator, the mortality is around 9 to 10 per cent. or, on occasion, even much higher (Cotte, Brentano, Villard, Duclos, Boutin, Sanders, Tixier, Clavel, Chabannes and others), depending on the presence of complications and other factors. My own series of 649 unselected routine cases operated upon over a period of 22 years by the classical method (between 1910 and 1932) disclosed a mortality in 138 males of 16.8 per cent. and in 511 females of 9.4 per cent. There is a noteworthy augmentation of mortality in males as compared with females. This, Tixier attributes largely to anatomic difficulties encountered in the narrow thorax of the male as compared with the ample lower thoracic cage in the female. Be this as it may, mortality in males following gallbladder operations is definitely greater than in females. It is thus obvious that the mortality following classical surgical procedures on the gallbladder still deserve the surgeon's serious attention.

Immediate Causes of Death.—Stanton[43] discussing the causes of death following gallbladder operations, pointed out that the danger here mostly arises from causes directly associated with the surgical method employed, despite excellence of technic. He studied the causes of death in 400 cases operated on for biliary tract disease reported in the literature, and 100 others in local hospital records, in all, probably representing about 10,000 operations. He lists as the most important causes of death, peritonitis, shock, hemorrhage, pulmonary embolism, pneumonia and cholemia. The first four enumerated represent those which admittedly lead as the immediate causes of death and are fraught with danger.

A certain rôle must also be ascribed to certain extrinsic causes. Davis[44] is of the opinion that the most glaring fault in gallbladder surgery in the past has been the reckless use of gauze and cigarette drains. Furthermore, peritonitis, the most common cause of death in biliary tract surgery (in Enderlen and Hotz's statistics it was almost double that of the next highest cause, pneumonia) is due to seepage of bile. The comparative merits of cholecystectomy and cholecystostomy is still a moot question; the literature is top heavy with divergence of opinions. I have watched the pendulum swing to the pro and con side of the question for over thirty years. The consensus of opinion today, however, is in favor of cholecystectomy, cholecystostomy being reserved to cases presenting strictest indication for its performance, and as shown above the mortality in cholecystostomy is decidedly higher than in cholecystectomy.

Drainage.—While the question of drainage following classical cholecystectomy has apparently a great deal to do with the question of complications and mortality, there are some surgeons who endeavor to close the abdomen without drainage, while others are still guided by Kehr's aphorism that "proper drainage is half a successful gallbladder operation." Paradoxical as it may seem, it nevertheless appears equally true that drainage is half an unsuccessful operation. No lesser surgical authorities than Witzel, Riedel, Rotter, Bier, Hoffmeister, Haidenhain, von Haberer, Ritter and others attempted series of classical gallbladder operations without drainage to return to the drain after sad experiences. On the other hand, some surgeons are of late again turning toward tight closure of the abdomen without drainage (Sanders, Verbrycke) (loc. cit.), Fowler[45], Richter, and Zimmerman[46] and others.

One may conclude that a dry operative field and a tightly closed abdomen seem unquestionably best, but with the present technic of scalpel cholecystectomy such ideal conditions and com-

plete satisfactory peritonealization cannot always be realized. Leaving speculative aspects pertaining to the causes of mortality following classical cholecystectomy out of consideration and limiting our analysis to factual desiderata, the subjoined premises are presented for consideration:

First: It is authoritatively pointed out that in normal individuals as well as in those coming under observation for disease of the gallbladder and extrahepatic biliary passages, the surgeon encounters in from 15 to 25 per cent. of cases, enlarged bile capillaries or small bile ducts coursing in the gallbladder bed (fig. 1). The surgeon performing classical cholecystectomy in these individuals divides the existing bile capillaries or bile ducts in the gallbladder bed with the scalpel, and bile leakage is inevitable.

Second: Bile not harboring pathogenic microorganisms will cause, when draining into the peritoneal cavity, a localized or even extensive chemical peritonitis while when infected, septic peritonitis eventuates.

Third: Leakage of bile from the cystic duct is too frequently accused of being the determining factor for bile escape. This is erroneous. A properly ligated cystic duct should not give rise to bile leakage. Bile invasion of the peritoneal cavity from this source is an exceptional occurrence. It is from the gallbladder bed whence bile leakage issues. This has been ably demonstrated by Bakes,[47] who in 1928 observed bile seeping from the operative wound 230 times in 346 cases of simple uncomplicated cholecystectomy, performed expertly, by the classical method. It is striking how often the surgeon notes bile on the dressings following clean cholecystectomy. Bakes was impressed by the appearance of bile in the dressing "in nearly all cases." From these observations the conclusion seems justified that the cystic duct ligature has not failed and that bile leakage resulted from the gallbladder bed.

Fourth: The majority of surgeons resort to drainage following classical cholecystectomy. Experience has taught them the inadvisability of primary closure of the abdominal wound following the removal of the gallbladder by the scalpel method. True, in many cases where the gallbladder bed is sutured and no drain is inserted, patients experience no particular postoperative trouble. Nevertheless, many sad experiences pressed the drain into the hand of most experienced surgeons anew. The late Lord Moynihan so aptly stated, that he always used a "conscience drain" in all cases. While omitting drainage following classical cholecystectomy may, on occasion, be condoned where a perfectly dry operative field pertains, one must nevertheless admit that in a great many cases of cholecystectomy the gallbladder bed cannot be obliterated by sutures; drains are then introduced under these circumstances and bile leakage and its consequences inevitably result.

Fifth: Not only is the exposed gallbladder bed a fertile source of bile leakage but in detaching the gallbladder, vascular spaces are also simultaneously opened. Blood seepage or even frank bleeding from vascular ostia predisposes to the development of thrombi which, on occasion may act as emboli followed by grave consequences. Also, such vascular openings may act as atria for the ingress of pathogenic microorganisms and be followed by serious infections. In other words, an open gallbladder bed is a gaping source of danger beset with the possibility of a variety of complications.

Sixth: The evils of drainage are well known. Foremost among these are augmentation of biliary and sanguinous drainage; thrombosis or embolism; hemorrhage from eroded vessels; vomiting due to duodenal compression; crippling adhesions; biliary fistulas; embarrassed cardiopulmonary action, etc. Or, to put it differently, many patients die after gallbladder operations not because they were not drained but because of drainage. Nevertheless, it must be remembered that in classical cholecystectomy, drainage is in principle, a *conditio sine qua non* and on occasion, life-saving.

With these facts before us, the question imposed was: *Supposing an operation could be evolved where the gallbladder bed was not disturbed by the operative procedure, would not such conditions add to the possibility of avoiding the evils of classical cholecystectomy and thus lower mortality? Supposing further, that a dry, sterile tampon of inert tissue could be substituted in what would otherwise probably be an open, gaping discharging wound, would such conditions tend for better results? Furthermore, since it is now recognized that cholecystostomy gives rise to a decidedly higher mortality than cholecystectomy, would not an effective operation*

instead of cholecystostomy be a step in the right direction?

To shed some light on the problems involved, our objective was to attempt to answer these questions through experimental and clinical researches. We proceeded as follows:

Fig. 1. Bile ducts in the gallbladder bed; magnified 19 times.

(A) An attempt was made to ascertain the reasons for clinical failures resulting from fulguration of the mucous membrane of the gallbladder (Pribram's so-called *mucoclasis*).

(B) Study the effects of electrocoagulation of the entire gallbladder wall.

The method of approach consisted of using dogs as experimental animals at first; later, *macacus rhesus* and *macacus leoninus* and finally applying the results of laboratory investigations to clinical evaluation.

Carbonization of the Mucous Membrane of the Gallbladder

In 1928, Pribram[48] described a procedure of destroying the mucous membrane of the gallbladder by cauterization. He called this mucoclasis (breaking up of the mucous membrane). Having used Pribram's method clinically, with disappointing results, I sought to ascertain the reasons for these failures. The technic described by Pribram was followed meticulously. After some weeks or months the gallbladders of the animals thus operated on were removed and examined histologically. Microscopic examination revealed that destruction of the mucosa and even portions of the muscularis by carbonization (to the limited depths which carbonization allows) acted only superficially. In patients the Rokitansky-Aschoff sinuses remained and infection persisted in the depths of the gallbladder walls thus treated and in some other clinical cases the mucosa completely regenerated.

This may be readily understood when we recall

(a) The anatomic structure of the gallbladder; and

(b) The limited surface effect of carbonization, fulguration or charring by any method, be it cautery or electrode.

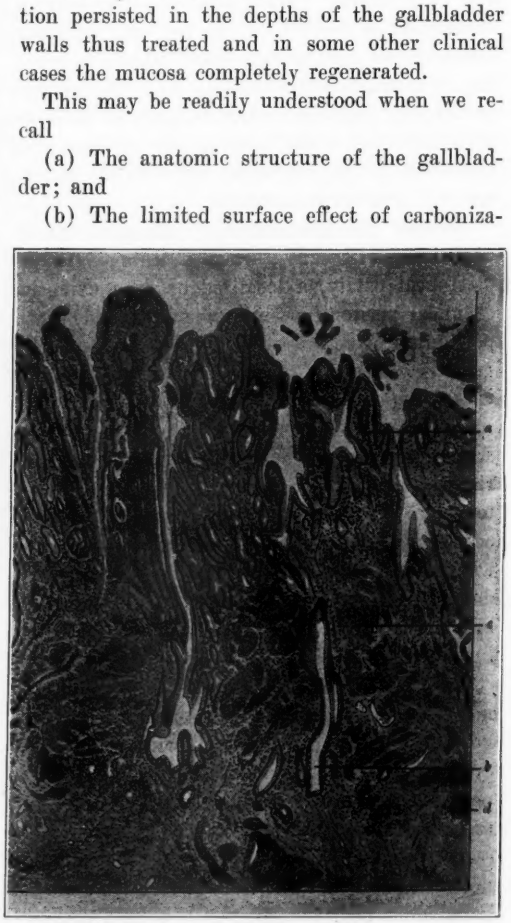

Fig. 2. Chronic hyperplastic cholecystitis. 1. (Roussy, Leroux and Oberling). a. Hyperplastic mucosa. b. Luschka's gland in longitudinal section. c. Muscularis. d. Inflammatory focus.

(a) *Anatomy.*—The gallbladder is composed of a mucous membrane which forms anastomosing folds and is lined with high columnar epithelium which secretes a mucoid substance

(globlet cells with basophil and exophil granules). It rests on a well developed basement membrane. The tunica propria contains collagen, elastic fibres and many lymphocytes, plasma cells and wandering cells. The muscularis consists chiefly of circular and a few oblique and longitudinal muscular fibres. The connective tissue membrane is divided into a fibrous layer of collagen fibres, a subserous layer of loose connective tissue and peritoneum.

The epithelium forms deep excavations with the underlying tissues which may perforate the muscularis and even reach down to the fibrous layer. These are known as the Rokitansky-Aschoff's sinuses described in 1842 by Piette.[49] They are also referred to as the glands of Luschka. According to Boyd[50] in chronically inflamed gallbladders these herniations or outpouchings of the mucosa may often be seen to pass for various distances into the muscularis and sometimes even into the perimuscular layer.

Limited Surface Action of Pribram's Mucoclasis.—I was able to show in 1933[51] and again in 1934[52] and 1935[53] and 1937[54] *that carbonization or fulguration by any method (cautery or electric current) will affect the mucous membrane but will not destroy the deeper structures composing the gallbladder wall, and that such limited destruction of the mucous membrane or even the muscularis for a limited distance will not destroy the deeper sinuses which may and in many instances, do, harbor pathogenic microorganisms* (fig. 2-6). The lymph apparatus of the gallbladder runs to the periphery of the organ and thence to the cystic gland. Infections may, therefore, be transmitted from the remaining sinuses to the liver and pancreas, again emphasizing the inadequacy of "charring" the mucous membrane (mucoclasis) in the hope of eradicating deep seated foci. It must also be recalled that besides the limited penetrating powers of charring produced by any agent, be it the actual cautery, electrocautery or by means of a diatherm apparatus, action is arrested as soon as an eschar is formed; this acts as an insulator and prevents further current diffusion to the deeper tissue layers. Particularly in gallbladders much thickened by disease, cauterization (carbonization) is very limited in action, affecting only the superficial portion (mucous membrane) of the thickened gallbladder wall. *These observations furnished the answer for the disappointing results of mucoclasis and why the procedure is ineffectual to eradicate gallbladder disease. It was obvious that Pribram's insistence on superficial action was responsible for the failures.*

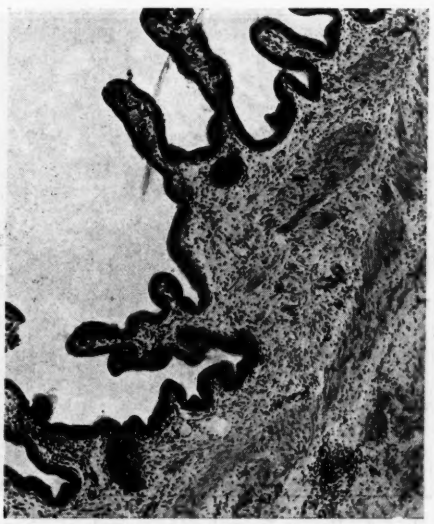

Fig. 3. Reason for failure of mucoclasis. Chronic cholecystitis nine months after electro-carbonization of mucous membrane of the gallbladder (mucoclasis). No relief of symptoms. Regeneration of mucous membrane and persistence of inflammation (round-cell infiltration, etc.). Relieved by electrocoagulation of entire gallbladder wall. (x 120).

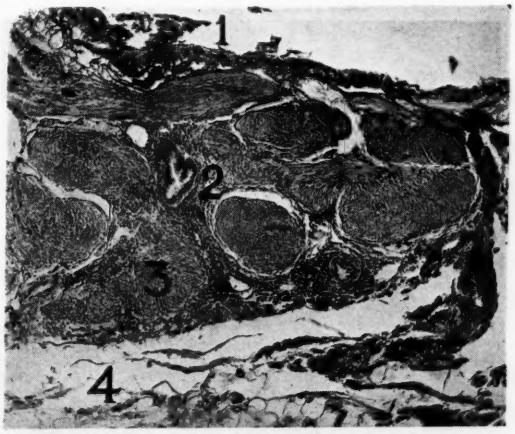

Fig. 4. (85). Effects of forced carbonization on wall of inflamed gallbladder. (1) Eschar. (2) Rokitansky-Aschoff sinus in transverse section. (3) Muscularis. (4) Serosa. Note excellent state of preservation of layers of gallbladder past the mucosa.

It must also be remembered that the mucosa may be normal and the mucoclasis harboring microorganisms.

After an animated discussion following his address before the Surgical Society of Berlin, Pribram stated that he borrowed the idea of charring the mucous membrane of the gallbladder from other sources. Thus Pribram wrote in the Zentralblatt für Chirurgie (loc. cit.):

Fig. 5. Reason for failure of mucoclasis. Chronic cholecystitis eight months after Pribram's mucoclasis (electro-carbonization) of the mucosa. Note remnants of Rokitansky-Aschoff sinuses and diffuse round-cell infiltration. Below, in center of field, an aggregation of polymorphonuclear leukocytes. Persistence of symptoms. Relieved by total obliteration of gallbladder wall by electrocoagulation. (x 65).

"It is a fact that partial burning of the mucous membrane of the gallbladder with the Paquelin cautery has formerly been practiced by various surgeons in order to avoid the formation of fistulas." From this, it is evident that "charring" and "carbonizing" of the mucous membrane of the gallbladder is not a new procedure but an old method adopted by Pribram from others which he christened "mucoclasis." Pribram further states: "Originally, I used the ordinary Paquelin burner to 'carbonize' the mucous membrane of the gallbladder. However, it was insufficient in action. I, therefore, used Hadenfeld's 'hot iron' such as was used in Bier's clinic. *Its burning (sic) effects were excellent* but it had two drawbacks: first, it produced too much heat in the abdominal cavity. Second, I was fearful of explosion while giving ether. I, therefore, used a diatherm apparatus known as the Penetrotherm. *This apparatus chars nad carbonizes excellently.*" Again in 1929 Pribram wrote[55] "*I want to point out that only superficial action is desirable, and, therefore, the electrode should barely touch the mucous membrane and not be pressed against the mucosa; if superficially touched small sparks will be seen to fly which destroy the mucous membrane at once.*"

Pribram continued his superficial charring method unmodified. As late as 1933[56] at the International Congress of Lithiasis he stated: "The principles of my method which I call 'mucoclasis' consist of burning the mucous membrane with an electric current. The burnt crust which results can be scraped off with a curette," and even at a later date, 1935, Pribram spoke of "burning," "charring" and "scraping."

A method was then sought to effectually destroy the entire gallbladder wall. Such a method was found in

(B) *ELECTROCOAGULATION OF THE ENTIRE GALLBLADDER WALL (Author's Operation)*

While Zschau[57] and Kuntzen and Vogel

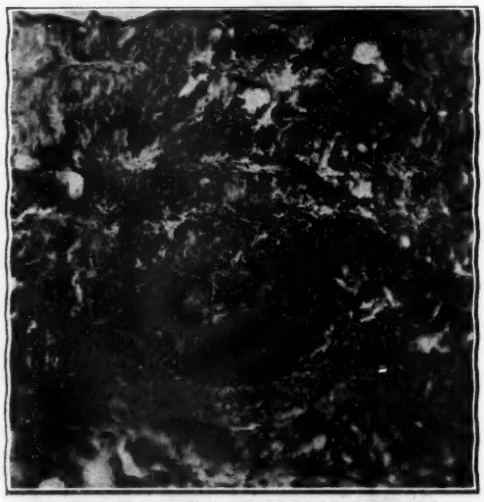

Fig. 6. Reason for failure of mucoclasis. Note area of round-cell infiltration in the wall of the gallbladder after Pribram's operation.

studied and described the effects of electrocoagulation on normal gallbladders *I have shown that by close contact coagulation one may con-*

vert the entire gallbladder wall into a hyaline-like, sterile, indifferent, dry tissue tampon (fig. 7). *Also, that if an electrode is used for fulguration, carbonization will result and the coagulating effect in the deeper parts of the gallbladder nullified.*

To fully comprehend the differences in the results between fulguration, cauterization, carbonization and electrocoagulation, the following should be recalled:

If a flat electrode is firmly applied to a tissue surface and a current of sufficient amperage permitted to pass through it, coagulation of tissue proteins results in a few seconds. This is shown by the white color assumed by the tissue thus treated. This represents electrocoagulation. On the other hand, if the electrode is not applied firmly, an air space (dielectricum) forms between the electrode and tissue to be acted upon, sparking (fulguration) results and carbonization characterized by a black discoloration of the tissues takes place. A reapplication of the electrode to such carbonized surface acts as an insulating surface and prevent further current penetration (fig. 8). Heat produced by cauterization is brought to the tissues from without; electrocoagulation produces heat in the tissues themselves, penetration is deep and the heat generated extends along the fluid conducting paths of the blood and lymph structures. In electrocoagulation the heat is produced in the cellular elements

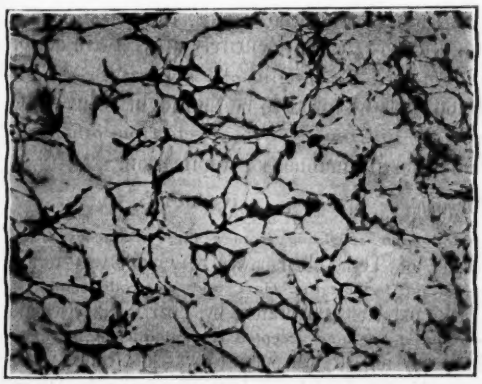

Fig. 7. Electrocoagulated gallbladder wall transformed into a hyaline-like network of indifferent tissue. (High magnification).

as a result of from 75,000 to 3,000,000 or more oscillations per second, depending upon the type of apparatus used (short wave).

Injections of India ink beneath Glisson's capsule of experimental animals showed that the action of the electrocoagulating current prevented the India ink from penetrating the tissues transformed by the current into a hemo-

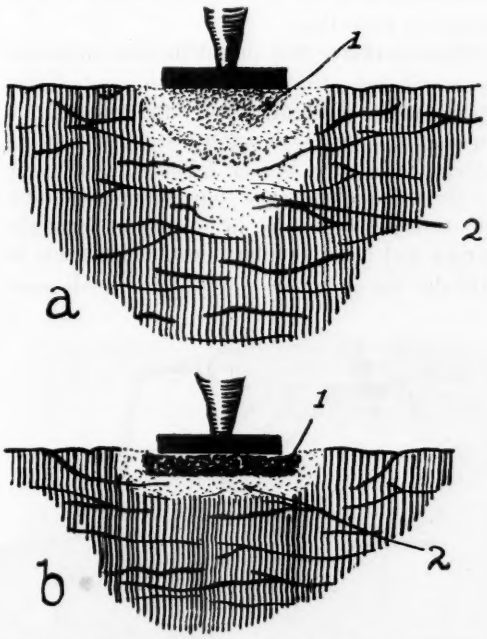

Fig. 8. Diagram of effects of *electrocoagulation* and of *carbonization*. In (a) one observes the degree and extent of electrocoagulation when correct technic is used. The dense area immediately under the electrode indicates that here the action is of greatest intensity. It gradually decreases as indicated in the diagram. Marginal action is seen to extend for some distance from the border of the electrode. When improperly used and too much current is applied charring results immediately under the electrode (b). Such eschar insulates the area acted upon and further effects of coagulation are frustrated.

geneous sterile structure. Zschaus' illuminating studies (loc. cit.) have established the remarkable effects of electrocoagulation on the blood and lymph vessels, showing that electrocoagulation does not cause thrombosis in the blood capillaries and smaller blood vessels. Occlusion of capillary spaces and vessels in electrocoagulated zones results from fusion of the electrocoagulated vessel wall structures. Such fusion is preceded or accompanied by a recession of the vascular contents at the point of contact with the electrode. The ends of the vessels become spindle shaped and fuse. All cellular structures, blood vessel and capillary elements are transformed into homogeneous tissue by coalescence and all capillary

and intercellular spaces are obliterated. Or as Ellis[58] says: "Hemostasis as a result of diathermy does not take place by thrombosis but by collapse and fusion of the walls of the vessels." Aschoff demonstrated that coagulation thrombosis is in sharp contradistinction to true thrombus formation.

While it is true that violent thermic influences (cauterization, etc.) may also close intercellular spaces where the heat contacts the tissues, it is equally true that such agglutination, limited in extent, to be sure, is soon followed, as a result of the marked hyperemia immediately adjoining the carbonized area, by an intensification of the lymph and blood capillary flow. This tends to dislodge the eschar and thrombi and predisposes

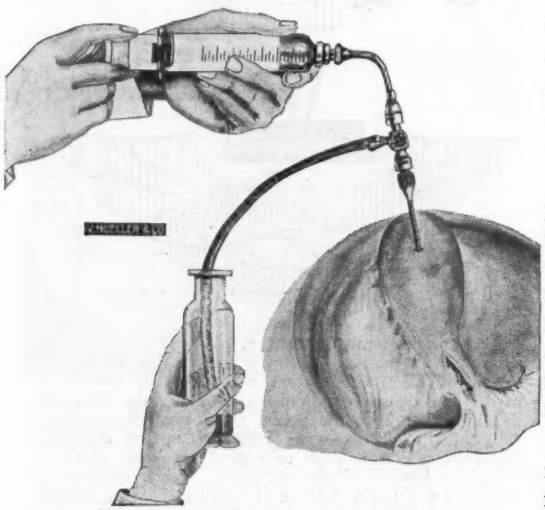

Fig. 9. Author's gallbladder aspirator.

to bleeding. This augmentation of wound secretion, consisting of lymph, toxic products, bacteria, loosened cellular elements, etc., may be responsible for considerable trouble. Electrocoagulation prevents this. In infected areas particularly, electrosurgery is of marked service.

From the foregoing description of its action, it can readily be seen that *electrocoagulation frustrates what carbonization encourages.* Electrocoagulation prevents formation and dissemination of thrombi and infectious material. If properly applied it avoids carbonization and thus eliminates its accompanying evils. The incidence of infarction pneumonias as a result of embolism is minimized.

Lack of absorption from electrocoagulated surfaces explains the absence of shock. Likewise, shock, as pointed out by Kobak[59] is prevented by capping the nerve ends during the coagulation process. Schörcher's[60] observations in this connection are illuminating.

It must be recalled here that electrocoagulated surfaces in various body orifices (mouth, vagina, rectum) heal by extrusion (sloughing) and final cicatrization; this is also true on cauterized surfaces in any part of the body. Intra-abdominal parenchymatous organs (liver, kidney, spleen) treated by electrocoagulation, on the other hand, tend to heal by encapsulation and final resorption. As long as there remains sterile tissues available to cover the electrocoagulated area, union and wound healing is not interfered with; even if only skin and subcutis are united over the electrocoagulated region the wound will still heal as long as no infectious material reaches the operative field from without or from within (von Seemen).

Electrocoagulated surfaces are possessed of a positive attraction for serous surfaces with which they become promptly agglutinated. They attract serous covered contiguous organs, in the form of a positive chemiotaxis, and become intimately united with these. I have observed agglutination three hours after electrocoagulating the surface of the liver. Firm union results in a comparatively short time.

Unfortunately, electrocoagulation is still confused by the uninitiated with cauterization and fulguration; these are entirely different in action and effects. To intelligently apply electrocoagulation, it is essential to be thoroughly conversant with the differences of its effects from other currents and its manner of application.

Terminology:

Monopolar sparkling from a high frequency transformer of Tesla, d'Arsonval, or Oudin was termed sideration by Keating-Hart and electrodissection by Clark, and later fulguration by Pozzi. Nagelschmidt introduced the now accepted term diathermy. Bipolar diathermy for surgical purposes was first called voltaization bipolaire and later electrocoagulation by Doyen; Delherme and Laquerriere spoke of endothermy showing that the heat is produced in the patient's body. George Wyeth in 1926, simplified the terminology by coining the terms medical and surgical diathermy. Henschen speaks of exsiccation, charring; Heitz-Boyer of etincelage; Cushing of dehydration; Kowarchik of electrotomy; von Seemen of Schmelzschnitt.

Historical

Priority for introducing electrosurgery in gallbladder disease belongs to Trowbridge. He described electrosurgical cholecystectomy with recovery, in 1928. Heymann (1930) substituted the diatherm knife for the scalpel, as did also Keysser, Mirizzi, and others; the results were little different from those of classical cholecystectomy. Pribram (1928, 1929) used carbonization of the mucosa with the Paquelin burner and later (1933) with a diatherm apparatus. Whitaker[61] in 1930 combined Pribram's cauterization procedure with drainage in 16 cases, disregarding the admonition that electrocoagulated surfaces must not be drained, and reported a mortality of 18.75 per cent.!

Following the publication of the results of my experimental researches in the ILLINOIS MEDICAL JOURNAL (November, 1933) pointing out the reasons for failures resulting from simple destruction of the mucous membrane of the gallbladder by mucoclasis, Pribram wrote: *"Thorek aims, if I understand him rightly, to destroy all the infected parts, the residual infection being one of the main causes of recurrent troubles after operation. . . . For this reason, Thorek insists upon total electrocoagulation of the gallbladder and its bed for the purpose of destroying deep-seated foci of infection. . . . Thorek's conclusions seem logical." In a later article*[62] in 1936, Pribram writes: *"At all events, Thorek's experiments have definitely proven that in operations on the gallbladder by electrosurgical means, the healing process, particularly in infected cases, is much improved."* In this connection, it is proper to quote L. R. Whitaker[63] who stresses: *"The method of Thorek emphasizing contact coagulation to considerable depths seems preferable to fulguration. I am grateful to Thorek for the distinction and wish to emphasize the necessity of real contact coagulation." Recently, Pribram*[64] *abandoned his ancient empirical method of "fulguration" and "burning" and has adopted the principal steps of my operation to be described below and he aims at the principle I have evolved, "at a dry, electrocoagulated surface."*

Our experimental and clinical studies have shown that:

1. The posterior gallbladder wall and bed of the gallbladder may be effectually electrocoagulated in toto and their component histologic structure converted into an inert dehydrated, hyaline-like, aseptic tissue tampon.

2. The electrocoagulated borders of the gallbladder thus treated may be easily approximated by means of sutures.

3. The falciform ligament may be completely detached and it, or a segment of omentum, superimposed over an electrocoagulated surface; these will unite intimately with the latter.

4. After electrocoagulation and approximation of the borders of the gallbladder and superimposition of the detached falciform ligament or a segment of omentum, the abdomen may be closed securely without apprehension, provided,

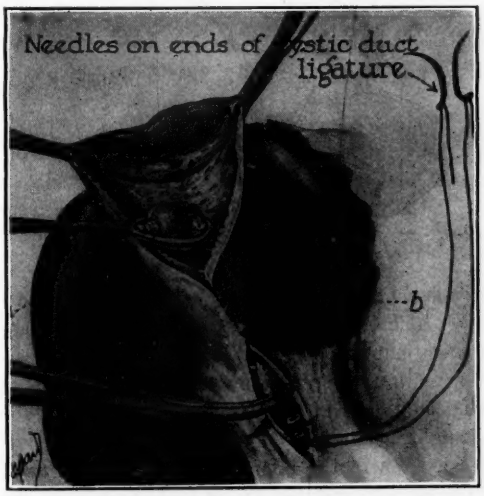

Fig. 10. After aspiration of the gallbladder the cystic duct and cystic artery are doubly ligated, the duct and artery divided and the ligature cut short (Author's operation).

the cystic duct is properly ligated and the cystic artery closed by double ligation.

5. Substitution of a sterile, dehydrated, inert tissue tampon represented by the electrocoagulated posterior gallbladder wall, instead of a bile and blood leaking surface often found in the gallbladder bed following classical cholecystectomy.

6. Strict avoidance of drainage.

Based on these observations, it seemed logical to expect that complete destruction of the posterior gallbladder wall by proper electrocoagulation would, perhaps, yield better clinical results. The following technic was then evolved.

AUTHOR'S OPERATIVE TECHNIC

Proper pre-operative care is, of course, essential.

Step 1.—Anesthesia and Position. In well trained hands, subarachnoid block is the anesthetic of choice. Charles H. Arnold is partial

Fig. 11. Author's non-spilling bile receptacle.

to splanchnic block in appropriate cases. Otherwise, inhalation anesthesia is used, in which case the patient is placed in the Mayo-Robson position with the lower thorax and upper abdomen sufficiently elevated; in subarachnoid block the straight position with upper abdomen raised is used. Where the biterminal apparatus is used, a large flat, indifferent electrode is snugly applied over the sacrum of the patient. If a uniterminal short wave apparatus is used an indifferent electrode is, of course, unnecessary.

Step 2.—Incision. A straight, longitudinal, paramedian incision is made beginning at the lower costal margin and extending to about the level of the umbilicus. Hamilton Bailey's transverse incision affords excellent exposure.[65]

Step 3.—Mobilization of the Falciform Ligament. This structure is completely detached from its insertion to the anterior abdominal wall. The detached ligament is now placed in a receptacle containing warm normal salt solution. If the falciform ligament is too small and cannot be utilized, an appropriate sized segment of omentum is resected and used, as described below, instead of the ligament.[66] In detaching the falciform ligament some small vessels are severed. These must be carefully ligated. Pack the viscera carefully away from the operative field and obtain good exposure by efficient retraction. A hypodermic injection of an ampule of prostigmine (1:4000), Hoffman-La Roche, before the operation is begun, aids in keeping the bowels contracted and out of the way. The use of prostigmine methylsulphate as an adjuvant during the operative procedure and postoperatively serves well.

Step. 4.—Exploration of the biliary passages. Digital exploration of the extrahepatic biliary passages is next done. The common duct is explored (instrumentally only, where indicated). The gallbladder is aspirated. The author's gallbladder aspirator permits evacuation of the gallbladder contents into a receptacle via a rubber tube (fig. 9). After thorough aspiration, the gallbladder is filled, through the same syringe with some antiseptic solution such as hexylresorcinol (S. T. 37) or other suitable germicide. It is well to recall that only about 5 per cent. of bile contains pathogenic microorganisms. On the other hand, the gallbladder wall harbors microorganisms in over 70 per cent. of cases.

Step 5.—Double ligation of the cystic duct and artery. In so doing elevate Hartmann's pouch with a suitable forceps. This facilitates identification and dissection of the ducts and ves-

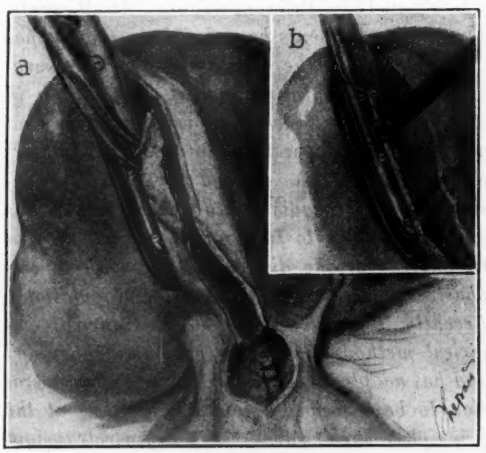

Fig. 12. (a) Opened gallbladder crushed with author's insulated angiotribe forceps and redundant portion of gallbladder removed with ordinary scissors. (b) Remaining strip of gallbladder in grasp of forceps being electrocoagulated with short-wave current. The current passes through the forceps but not beyond the insulated protector (Author's operation).

sels. I prefer to ligate the cystic duct and artery jointly instead of resorting to extensive dissections, particularly in a much infiltrated hepatoduodenal ligament. The frequently abnormal course of the cystic artery should be kept in mind. Duct anomalies should also be remembered (fig. 10).

Step 6.—Open the gallbladder and evacuate its contents by splitting it with a straight pair of scissors from above downward. The author's gallbladder content receptacle collects the evacuated material without spilling it (fig. 11). Old fashioned gallbladder spoons are too shallow and offer no protection against contamination from spilled contents. My instrument consists of a cone-shaped container measuring 2¼ inches in diameter and 2 inches in depth, mounted on a shaft of suitable size. A close fitting cover is hinged and held open when in use by means of spring friction clips; slight pressure upon the cover releases it so that the material cannot spill during its removal. The front part of the receptacle is somewhat concave allowing sufficient latitude for proper contact with the subcholecystic structures, and its edges are rodded to protect the liver from injury.

Step 7.—Grasp half of the gallbladder wall with an insulated angiotribe forceps or a straight rubber covered crushing forceps of proper size and thickness. In closing the forceps, the gallbladder wall is crushed and its blood vessels closed by compression. With an ordinary pair of scissors, remove the redundant portion of the pathologic gallbladder wall (fig. 12-a).

Note: In some instances, crushing with an ordinary forceps and cutting off the affected gallbladder wall along the forceps leaving a small projection of gallbladder tissue and electrocoagulating same, will serve the purpose.

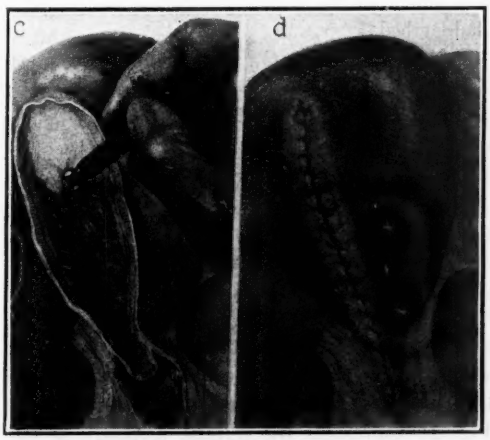

Fig. 13. (c) The insulated angiotribe forceps are removed leaving ribbon of crushed and electrocoagulated strip of gallbladder tissue all around gallbladder bed. The posterior wall of the gallbladder in the middle of this ring is being electrocoagulated. (d) Strip of electrocoagulated and crushed ring of tissue of gallbladder united with continuous catgut sutures (Author's operation).

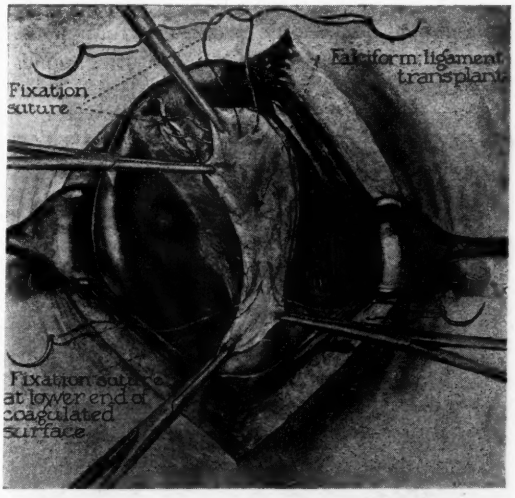

Fig. 14. Note borders of the electrocoagulated area approximated with fixation suture. Additional interrupted sutures of fine catgut approximate the borders of the electrocoagulated zone along its entire extent. The mobilized falciform ligament is fixed over the closed, electrocoagulated surface. Note placement of sutures (Author's operation).

Step 8.—Apply a small electrode firmly along the exposed margin of the gallbladder wall held in the angiotribe or crushing forceps. The smaller the electrode, the quicker the action. The current will coagulate the gallbladder wall held in the grasp of the forceps. After sufficient coagulation, the forceps is opened and the released margin of the gallbladder wall will appear as a compressed ribbon of yellowish tissue (fig. 12-b). Repeat the procedure on the other half of the gallbladder.

Step 9.—The remaining portion of the posterior gallbladder wall remains now to be thoroughly coagulated (fig. 13-c). In my early work, I thought it advisable to electrocoagulate slowly but experience has shown that quick coagulation with firm electrode contact yields the best results. I use a current of low voltage and high amperage through a small electrode. On

this unit (Fischer Model S. W. 12) as the voltage drops the amperage increases; this affords prompt, thorough dehydration and a sharply defined line of demarcation between the coagulated and non-coagulated tissue. In this manner, less moisture is retained in the tissues than when coagulation is done slowly. In short and decisive coagulation, less heat is radiated to contiguous tissues. Another reason for substituting rapid coagulation with a smaller electrode is that slow

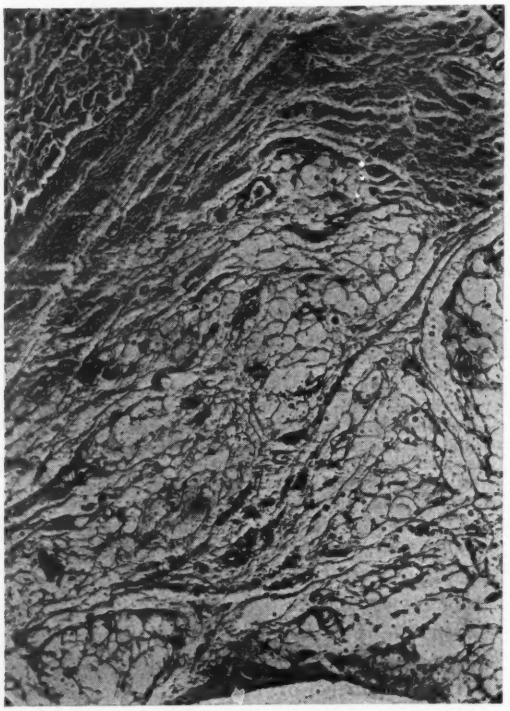

Fig. 15. Transplanted and completely detached falciform ligament eighteen months after electrosurgical obliteration of the gallbladder. Observe normal histologic appearance of liver structure. All that remains of the gallbladder bed is a strip of connective tissue. The transplanted falciform ligament shows its characteristic normal structure (Low magnification).

coagulation results in heating of the tissues to quite an extent beyond the electrode contact, about 0.5 cm. for each mm. of coagulation. In slow coagulation, there results no sharp line of demarcation—the tissue changes are gradual.

It must be emphasized that the electrode must be applied snugly in order to avoid carbonization. The current should be turned on only after the electrode is firmly in contact with the tissues to be acted upon and turned off before contact is interrupted. *Fulguration and its attendant carbonization must be avoided because the latter defeats the purpose of the operation.*

When the steps of the operation are carried out as here outlined, the operative area will be represented by the electrocoagulated posterior wall of the gallbladder attached to the gallbladder bed.

Step 10.—Approximate the electrocoagulated edges of the gallbladder by interrupted sutures of either catgut or fine silk or linen. During the approximation of the electrocoagulated edges the needles must not be passed through adjacent liver substance; only the edges of the coagulated ribbon should be picked up (fig. 13-d). Continuous sutures are only resorted to where catgut is used.

Step 11.—The detached falciform ligament or free omental graft is now removed from the warm salt solution. The upper end of the ligament is sutured to the superior margin of the now coagulated and united gallbladder bed (fig. 14). While the lower end of the ligament or graft is stitched to the lowermost end of the approximated electrocoagulated surface; the free end of the ligament is permitted to hang over the ligated cystic duct area to which it is *not* sutured. An effective serous covering is thus provided over the united gallbladder bed and cystic duct area. As a rule, only two sutures are required to accomplish the attachment of the serous covering over the gallbladder bed.

What becomes of the transplanted falciform ligament or omental graft after superimposition on the gallbladder bed? Animal experiments and clinical results furnished the answer to this query. A postmortem examination was done on a patient who died of acute pulmonary tuberculosis on whom I performed electrosurgical obliteration of the gallbladder eighteen months prior to her last illness. Fig. 15 depicts the histologic structures. Figs. 16 and 17 show the histology of the gallbladder bed from a patient who expired on the day of her discharge from the hospital from pulmonary embolism, while Fig. 18 represents the minute anatomy of the transplanted area ten months after operation. The specimen was obtained with the consent of the patient during an operation unrelated to gallbladder disease.

Step 12.—Remove the laparotomy packs and retractors; lower the bridge of the operating ta-

ble thus relaxing the abdominal wall to facilitate closure; close the abdominal wall in layers without resorting to drainage.

RESULTS

With Pribram's mucoclasis: Clinical failures: the reasons for these have been described and are borne out by microscopic studies (see fig. 3-6).

With Author's Operation: Up to the present writing (September, 1940) there were performed 980 operations of electrosurgical obliteration of the gallbladder with a total mortality of 0.3 per cent.

Bailey and Love	152 cases—no mortality
Roman, Kellogg, Warren, Finalyson, Baller, de Victoria, Stevenson, Weyman, Heineck, Lapenta, Arnold, Best, A. S. Jackson, Pobe, Behrend, Kiesser, Wise, Watts, Palmer and Montgomery	336 cases—no mortality
Author	491 cases—0.5% mortality
Total	980 cases

Bailey and Love*[67] summarize their observations as follows:

"The following are the results obtained in our series of cases by the employment of Thorek's Method:

No. of cases	129
Healed by first intention and uneventful recovery	122
No. of cases of complications	7
Average stay in hospital	20 days
Mortality	Nil

In the Author's series of cases the following observations were recorded:

Ratio: One male to six females.

Age: Youngest, 8 years; oldest, 74 years.

Average high temperature, 100° F.

Average days in hospital, 15.5.

Pathologic conditions found at operations—(1) Acute and chronic forms of cholecystitis; (2) cholelithiasis; (3) hydrops vesicae fellae; (4) empyema of the gallbladder; (5) acute hemorrhagic cholecystitis; (6) hepatitis; (7) adhesions; (8) chronic pancreatitis. Postoperative complications—(1) bilateral pleurisy with effusion, ten days after operation; cleared up; patient left hospital 27 days after operation; (2) wound infections—3 cases (about 0.5 per cent.).

Mortality—(four deaths in 980 cases equals less than one-third of one per cent.)

Causes of death—bilateral pulmonary collapse; pneumonia three weeks after operation; unrecognized chronic subdiaphragmatic abscess; pulmonary embolism; patient up and about but still in hospital.

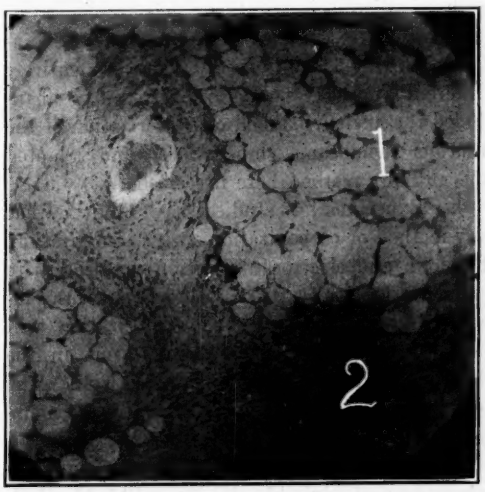

Fig. 16. Histology of gallbladder bed 12 days after operation. Section shows union of falciform ligament (1) to electrocoagulated gallbladder bed (2). The latter is represented by fibrous tissue.

SUMMARY AND CONCLUSIONS

1. A method of electrosurgical obliteration of the gallbladder is described which, when carefully followed, may be used in simple and complicated cases of gallbladder disease without resorting to drainage. It reduces mortality and cuts hospitalization. The much dreaded age factor in these operations loses much of its terror. Shock is usually absent

2. Failures and fatalities following classical cholecystectomy are frequently due to bile leakage, as a result of an inability to surgically obliterate the gallbladder bed, which contains bile capillaries and other larger bile ducts, in 15 to 25 per cent. of cases. Drains invite bile seepage; the method here described seals these openings by electrocoagulation. A sterile hyaline, dry

*Since writing this Hamilton Bailey states (personal communication) that he has performed 23 more operations by this method and reports no mortality.

The gallbladder is a distinct advance on the standard operation of cholecystectomy. The more important advantages are:

"Diminished mortality. Thorek found his mortality in 937 unselected cases to be 0.3 per cent. The average mortality of the standard operation in skilled hands is 2 per cent.

"Postoperative discomfort is reduced to a minimum. We have been deeply impressed by the smoothness of convalescence and absence of worry symptoms as compared with those which commonly occur during the first few days, after the standard procedure. Many patients have no more discomfort than if they had undergone a simple appendectomy. Rapid convalescence. It is exceptional for the wound to heal other than by first intention."

protective layer is substituted for a raw, unprotected gaping wound so often seen following classical cholecystectomy. The falciform ligament or a free omental graft is superimposed over this area. Drainage is rendered superfluous and is entirely omitted.

3. The operation here described must not be confused with Pribram's mucoclasis practiced with the cautery or by "diatherm-fulguration" which aims at "burning" or "carbonization" of the mucous membrane of the gallbladder wall or with so-called "electrical" or "electro-cholecystectomy" where, instead of the scalpel, the diatherm cutting knife is used. These are entirely different procedures. Pribram recently abandoned the ancient method of cauterization of the mucous membrane (mucoclasis) and adopted the author's method here described.

4. Experimental studies have shown that any method of carbonization such as fulguration, Paquelin and electrocauterization predisposes to eschar formation, extrusion, hemorrhage, thrombosis and embolism. Such cauterization will not destroy pathogenic microorganisms in the depths of the affected gallbladder wall. The operation here described is free from these drawbacks and effectually accomplishes destruction of the entire thickness of the gallbladder wall and where deemed advisable also the gallbladder bed. The surgeon has under control the degree of penetration he wishes to accomplish.

5. A prerequisite to the achievement of satisfactory results is a patent common duct. An occluded cystic duct from any cause is an indication for this operation.

6. I have shown that the ligamentum falciforme hepatis or a free omental segment is used as a completely detached graft—its serous structure is made to cover sutured or raw surfaces to great advantage, thus reinforcing and protecting the areas concerned against seepage and safeguarding the processes of repair. The transplanted falciform ligament or free omental graft becomes intimately united with the gallbladder bed area.

7. Electrocoagulated areas of intra-abdominal organs tend to heal by encapsulation. They do not interfere with wound healing. On the contrary, they heal promptly when the wound is closed securely. Therefore, drainage is not only undesirable but is distinctly deleterious. Many patients succumb because of drainage. The author's operation here described eliminates the necessity of drainage and its unpleasant sequelae.

8. The thesis here presented deals with diseases of the gallbladder. Concommittant affections of the extrahepatic biliary passages must be treated according to indications. Where choledochostomy or hepaticostomy is indicated, these may be done, in conjunction with electrosurgical obliteration of the gallbladder. Experimental studies showed that the implanted falciform ligament over the obliterated gallbladder bed forms sufficient protection and does not interfere with common or hepatic duct drainage.

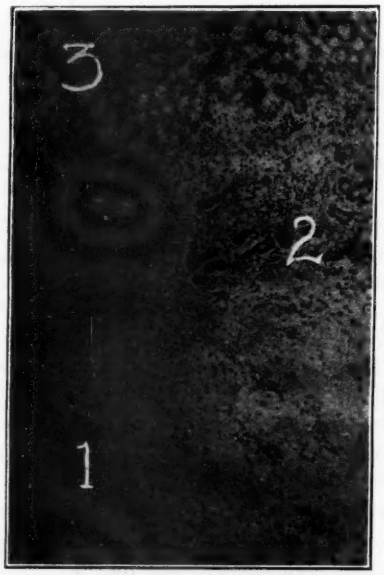

Fig. 17. Section taken through electrocoagulated posterior wall of the gallbladder (1) showing it being transformed into a hyaline line structure (2) compressing the gallbladder bed. (3) Histologically normal liver structure.

9. Inasmuch as the global mortality of removal of the gallbladder by the scalpel method in complicated cases still is considerable and since the series of 980 unselected cases here reported, this operative procedure resulted in the very low mortality of about one-third of one per cent., the effort of acquiring an exact technic for its performance seems worthwhile. Furthermore, since reports indicate that cholecystostomy results in a far greater percentage of deaths than cholecystectomy (vide supra), it is reasonable to substitute in many acute cases the

author's operation for cholecystostomy. In fact the author and others have consistently practiced this method in chronic as well as in acute cases.

10. Electrosurgical cholecystectomy—electrocholecystectomy—so-called, is an entirely different procedure and is nothing more than classical cholecystectomy where the electric cutting knife is substituted for the scalpel. It has contributed nothing to the improvement of the old method and has added the danger of explosion by sparking or fulguration. Furthermore, if coupled with drainage as practiced by Whitaker (loc. cit.)

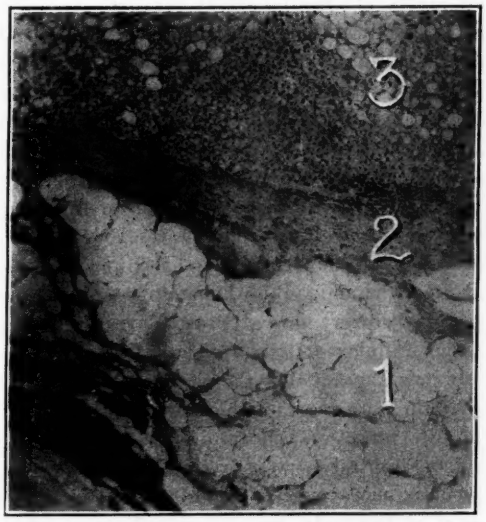

Fig. 18 Histologic structure of transplanted falciform ligament gallbladder bed and adjoining liver tissue ten months after electrosurgical obliteration of the galbladder. Note excellent histologic preservation of ligament. Gallbladder bed represented by fibrous band and normal histologic appearance of liver structure.

more drawbacks are added in the form of increased mortality (18.75 per cent.!).

11. Many decades ago a variety of attempts were made to destroy the mucous membrane of the gallbladder by carbolic acid and other escharotic substances or by the thermocautery or fulguration as practiced by Pribram. These are antiquated procedures, superficial and ineffective. Some demand prolonged periods of drainage and often leading to fistualization. Recurrences were noted in the majority of cases where mucoclasis was used.

12. Secondary hemorrhage from the gallbladder bed does not take place in a properly performed electrosurgical obliteration operation of the posterior gallbladder wall because of the tampon like pressure exerted by the coagulated tissue against the contiguous gallbladder bed.

13. A survey of mortality statistics and historical data is presented.[68, 69, 70, 71]

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APPLICATIONS OF VITAMIN B_1 TO NEUROPSYCHIATRY

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Neuropsychiatry is the first of the domains of medicine to be touched by a recognition, nebulous as it was, of vitamin deficiency. This happened long before the word was coined, long before these accessory food substances attained a fraction of the importance they now have. The Chinese wrote of beri beri centuries ago. Descriptions are mentioned as far back as 2,697 B. C. Clinical descriptions from the Orient and the East Indies of what we now call vitamin B deficiency antedate the appellation of "lime juicers" or "limeys" to British sailors who used this citrus fruit for prevention and treatment of what we now know as a vitamin C deficiency, scurvy. It remained, however, for an American worker, Robert R. Williams, a research chemist, to study first the structure and then to synthesize thiamin, vitamin B_1. Laboratory applications of this discovery have given us much information about cellular activity, for as McLester[1] says, "It now appears that the chief function of this vitamin is to regulate the most fundamental of all life processes, cell respiration." Since cells of the central nervous system require proportionally more oxygen than those of any other part of the body, since they suffer more from oxygen insufficiency than do other cells, since the integrative character of nervous activity shows up such an insufficiency in many ways the importance of thiamin is logical. Several disorders hitherto unexplained by infection, trauma and circulatory incapacity found explanation as a result of the discovery of the importance of B_1 for oxygen up take. It is the purpose of this paper to discuss some of these.

As indicated in the previous paragraph beri beri, particularly the dry or wasting form, was the first connotation of dietary factor in the development of systemic disorders. When McCollum introduced the alphabetical system of designating the substances Funk first called vitamins, vitamin B was classified as the one associated with this disorder. Various functions of import-

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ance have been assigned to certain fractions in the B complex of water soluble substances. The relationship of these is shown in the chart from the monograph of Williams and Spies.[2] Some of these are complementary to each other in their activity, some appear to have selectivity in certain conditions. Even before the complex was broken down into the fractions now known to exist, deficiency of vitamin B was linked with other disorders than beri beri. Schlesenger[3] in 1919 described cases of polyneuritis as occurring in hunger edema seen in Central Europe. McCollum[4] in an early edition of his book, "The Newer Knowledge of Nutrition," speaks of other disorders.

The writer's interest in vitamin B deficiency dates from the report of Williams on the structural formula and from a paper by Cowgill[5] in 1929 referring to work on vitamin B. Practical clinical results in disorders like pernicious anemia and diabetic neuritis reported by him stimulated personal observations in these and other situations where dietary elements could be considered as entering into neuropsychiatric conditions. Results were such as to justify the addition of foodstuffs high in vitamin B content such as yeast and malt extracts to the dietary of patients in the Sanatorium. Particularly were these additions given to those who through inability to cooperate could not partake of the standard high calory high vitamin general diet offered them. Cowgill[6] worked out the vitamin B requirement of man in dietaries. While these are in the main associated with beri beri one can figure from them the amount present. Certainly so far as the average American general diet or hospital diet is concerned requirements are adequately met. So there must be some other factors in the picture.

These factors appear to be (1) an "unaverage" diet of economic necessity, dietary fads or idiosyncracies; (2) difficulty in absorption or utilization of food by reason of ingestion of substances that either affect such ingestion or utilization, i.e., alcohol; (3) organic disease; (4) increased requirement not met by corresponding increase in proper food intake, i.e., pregnancy or lactation. One or more of these factors may work together as in the instance of an indigent pregnant woman or an alcoholic diabetic.

When thiamin chloride became available in larger amounts for experimental and clinical use tremendous impetus was given to studies in this field. Similar to the experience with insulin or liver extract the clinical applicability of thiamin chloride has been accompanied by extensive physiological studies. Insulin has given us practicing physicians an essential item in treatment of diabetes mellitus. It has also given us through physiological studies greater knowledge of carbohydrate metabolism. Liver extract has not only given us a specific for pernicious anemia but has also opened new fields of investigation of the hematopoietic system. Early clinical application of the use of vitamin B_1 in treatment of alcoholic neuritis has extended the scope of neurophysiology to include work that indicates factors in the metabolism of nerve tissue and of cellular respiration throughout the animal and vegetable kingdoms. We see advertisements of grass seed treated with vitamin B_1 so that our lawns should look like well kept greens. Yields of some crops are definitely increased.

We owe to Williams and his collaborators in this country the technique for the synthetic production of vitamin B_1. The procedures used are capable of control by the methods of organic chemistry. Manufacturers have taken these up so that active and accurately standardized preparations are now available.

The Council on Pharmacy and Chemistry of the American Medical Association has designated the term thiamin chloride as the official name of vitamin B_1. Various other names are applied by different manufacturers. It is often the case in medicine that enthusiasm for a new preparation leads to its utilization for many conditions. Where disorders of the nervous system are present one needs to be more than ordinarily careful in interpreting results. Emotional reactions, suggestibility and so-called functional elements have to be evaluated properly. Three groupings bave been chosen to indicate neuropsychiatric disorders in which vitamin B_1 (1) definitely aids, (2) apparently is of some value, (3) has been reported in isolated instances to be helpful.

TABLE 1

Neuropsychiatric disorders definitely aided by Vitamin B_1

- Polyneuritis due to dietary deficiency
  - Beri beri and allied conditions
  - Alcoholic polyneuritis
  - Other nutritional polyneuritides, dietary errors, diabetes, pernicious anemia, cachectic conditions

Polyneuritis due to excessive demands
Pregnancy
Lactation
Polyneuritis due to heavy metal intoxication (Source controlled)
Arsenic
Lead
Alcoholic encephalopathies
Korsakoff's psychosis
Delirium tremens
Alcoholic hallucinosis

As seen from this table various forms of polyneuritis are conditions in which there is definite proof of the value of vitamin B_1. Reference has already been made to beri beri. Deficiency in alcoholic polyneuritis has been shown to be due to two factors. First is loss of appetite which accompanies alcoholism, lessens intake of normal food elements in adequate amounts. Second is lessened absorption from irritated gastric mucosa. There is not conclusive evidence that alcohol in the tissues is a deciding factor though it is considered by some to participate in the demyelinating process. Therapeutic application of the above calls for three things, first and foremost stopping alcohol, second adequate diet, third thiamin chloride in dosage sufficient to overcome the deficiency. We give all alcoholics doses of thiamin chloride, 6,000 units or more intramuscularly, daily. Even though McGee[7] found practically no difference in time required for complete alleviation of symptoms between a diet-plus-thiamin-treated group and a diet-treated group we believe on the basis of present knowledge it is a help.

Many other types of nutrition polyneuritis come more within the scope of general medicine than neuropsychiatry. Because we do see personally a rather large number of diabetics we refer to diabetic neuritis. While many of the discomforts in the lower extremities in diabetes are due to vascular lesions of an arteriosclerotic nature careful neurologic examination will often reveal evidences of nerve involvement. Younger diabetics showing neurologic changes and practically all over fifty years of age now are given thiamin chloride with apparent relief of many of the symptoms. It is desired in this connection to raise the question whether insulin, a sulfur-containing hormone and thiamin, the only sulfur-containing vitamin thus far isolated, may not complement each other's activities.

As regards pernicious anemia neurologic symptoms are often far more disabling from the patient's standpoint than the anemic ones. That the anemia is the primary factor in these is now generally recognized. Associated with the development of the anemia either as part of the extrinsic dietary phase or the intrinsic inability to utilize food products there may be vitamin deficiency. Peripheral nerve changes are known to exist in pernicious anemia. Vitamin B_1 plus liver appears to be of benefit for these and perhaps for cord changes as well. According to Wechsler[8] the possibility that changes in subacute combined degeneration of pernicious anemia are due to a vitamin deficiency is the reason for vitamin therapy here. Our own experience has led us to a similar conclusion.

Polyneuritis of cachetic origin is well known. Anorexia, toxic and degenerative changes all contribute. Administration of thiamin will not, of course, affect a malignant process but where neuritic symptoms are metabolic and not of pressure origin, it will help them.

Our attention was called in 1936 to results in polyneuritis of pregnancy treated with vitamin B_1, by the paper of Sahs[9] at the Central Neuropsychiatric Association meeting. The results there reported indicate value in this condition where excessive demands may be hard to meet unless care is taken in the diet of the expectant mother. Wechsler[10] draws attention to the fact that in many cases of polyneuritis gravidarum there was a history of intractable or pernicious vomiting, hence starvation. Similar conditions apply to the nursing mother.

Heavy metal poisoning inducing neuritis affects the general metabolic processes of the body as well as producing changes in peripheral nerves. It goes without saying that the exogenous source of metallic poisoning needs to be controlled. When this is done administration of vitamin B_1 appears to expedite recovery.

Alcoholism produces central nervous system lesions as well as in the peripheral nerves. In fact in our experience at the Sanatorium delirium tremens is more common than alcoholic polyneuritis though symptoms or signs of neuritic change may be found in many alcoholics. Polyneuritis is considered as one of the diagnostic criteria of Korsakoff's psychosis along with confabulation or the filling in of lapses in the stream of thought and speech by imaginary speculations. Cases reported and our own experience indicate definite value of vitamin B_1 here. Joliffe[11] in a carefully controlled study

states that, "subjects receiving thiamin chloride showed an incidence of recovery seven times as great as those treated by diet alone." He adds, however, that "the variable factors in a disease like the Korsakoff psychosis, and the difference in length of observation, as well as the small size of the treatment groups when further divided into sub-groups of acute and insidious onset, make it difficult to draw definite conclusions concerning etiology or the effectiveness of the specific therapy."

The variety of methods used in the treatment of delirium tremens make it likewise difficult to evaluate results here. Gradual withdrawal of alcohol, abrupt cessation of it, spinal drainage, intravenous glucose, insulin and subconvulsive dosage of metrazol all have their advocates either used singly or in some combination. It is our impression, though we would not want to state it positively, that patients with delirium tremens appear to have less shock reaction to stopping alcohol since thiamin chloride has been added to our regime than was previously the case. We have been interested in the work of Robinson on the use of insulin in this situation and have again wondered if there was any connection between the activity of this sulfur containing hormone and the sulfur containing vitamin, thiamin.

Table 2 shows some neuropsychiatric disorders in which sufficient reports are at hand to indicate that vitamin B_1 may be helpful.

TABLE 2

Multiple sclerosis
Neuralgias
Ischemic neuritis
Insulin hypoglycemia
Nerve exhaustion syndromes (so-called, including hyperthyroidism)

We have been using thiamin chloride in a few cases of multiple sclerosis for the past two years. It was started on the reasoning that in a demyelinating lesion it should theoretically be helpful. This was before we read any reports of its use. While Mellanby has thought of vitamin A deficiency in multiple sclerosis, while there is good experimental evidence that such deficiency can produce degeneration of cells of the central nervous system, certain objections appear to us in the case of multiple sclerosis. Among these are the changes in the visual purple of the retina associated with night blindness in vitamin A deficiency in which so far as we know have not been reported in multiple sclerosis. Pathologic changes in the visual apparatus are retrobular neuritis, optic atrophy and nystagmus with third nerve nuclear involvement. Murphy has reported on several cases of multiple sclerosis treated with thiamin chloride and nicotinic acid.

Considerable publicity was given last year to the treatment of tic douloureux by thiamin. Our experience in two cases was disappointing. We would recommend the selective nerve operation proposed by Klemme rather than depend upon vitamin treatment for this disorder. Some mild neuralgic type situations have responded well to vitamin B_1 for use in controlled patients where no analgesic medication was employed, but as indicated previously in this paper one must keep one's fingers crossed when dealing with neuropsychiatric conditions. However, we consider thiamin an adjuvant treatment of some value in neuralgic disorders, including sciatica, and use it.

One type of ischemic neuritis has been mentioned in the discussion of diabetic neuritis. Peripheral vascular disease in non-diabetics also comes under this heading. Another one where we have thought it helped has been in two instances of Volkman's contracture which have come under observation. Anything that would promote metabolism of nerve tissue should be helpful under such conditions.

The use of vitamin B_1 in insulin hypoglycemia is pretty much self-limited to insulin shock therapy. Practically all insulin reactions in the treatment of diabetes mellitus are promptly controlled by easily absorbed carbohydrates such as orange juice or corn syrup given by mouth. Glucose solution given by mouth or nasal tube takes care of a large proportion of induced insulin shock in schizophrenia though at times it is necessary to give glucose intravenously. Freudenberg[12] has used thiamin chloride as an aid for patients who do not respond to this. Others report its use intraspinally as well. We are in entire agreement with the statement of Wechsler (ibid) that "the administration of vitamins by the intraspinal route is not justified on the basis of present knowledge" and that "there is no evidence at present that the vitamins can get past the barrier between the cerebrospinal fluid and nerve tissue."

An important contribution has been made by Checkley, Sydenstricker and Geeslin[13] in reporting good results with the use of thiamin chloride and nicotinic acid in some states of mental con-

fusion in elderly individuals. All physicians see patients belonging in this category. Cerebral arteriosclerosis with diminished blood supply to the brain interferes with normal metabolism of nerve tissue. Loss of appetite with resultant lessened food intake have parts in the picture too. Proper management with adequate food intake is undoubtedly the main factor in controlling such a situation. It has been our observation that in addition to these the use of vitamins is distinctly helpful, especially thiamin chloride and nicotinic acid.

There are other neuropsychiatric disorders with isolated reports of value of vitamin B_1. These are shown in table 3. Some seem to the writer at this time far fetched as to their applicability, Time and further studies under controlled conditions will render the decision as to presence or absence of value.

TABLE 3

Neuropsychiatric disorders with isolated reports of relief by Vitamin B_1.
Poliomyelitis
Encephalitis, acute and chronic
Parkinsonism (idiopathic and post encephalitic)
Progressive muscular atrophy
Myasthenia gravis

Discussion of the value of vitamin B_1 in other than neuropsychiatric disorders or those having important neuropsychiatric components does not come within the scope of this paper. Certain heart conditions, irradiation sickness, general asthenia, anorexia and growth discrepancies are important medical problems where this vitamin is quite definitely of value. As further research is carried on, the question, if there is associated nervous system involvement there, will be further worked out.

A few practical points in the use of vitamin B_1 are worthy of mention. Cost is a matter of concern to all but the rare patient. The husband of one of our out-patients could not be convinced that his wife needed or would be benefited by tablets that cost a nickel each. Yet if one is going to give oral medication the 3.3 milligram tablets which supply 1,000 international units are cheaper in the long run than giving more of a smaller size. The writer does not see value in the various vitamin B_1 elixirs which contain relatively little thiamin chloride yet which are much more expensive than simple elixir as a vehicle for other drugs. Hospitalized patients and those in office practice who are able to come in frequently will get more benefit and therefore more value received by parenteral injection of solutions of the strength of 50 milligrams or 15,000 units per cubic centimeter. The actual cost of 20 milligrams or 6,000 units of such a solution should be 28 to 48 cents, depending upon local conditions, and whether purchased by the physician or by the patient on prescription. Where facilities for preparation of sterile solutions are available as in central supply departments, pharmacies or laboratories of most hospitals and in some physician's offices cost can be cut still further. Some manufacturers sell the crystalline thiamin chloride which is readily made up in sterile solution of desired strength. This is the method we employ.

Vitamin B_1 is an important addition to the therapeutic armamentarium of the physician. It has significant value in some neuropsychiatric disorders, alone and in combination with other members of the vitamin B complex. It is essential in its use as in the use of any form of medicine to have an indication for employing it. Such an indication includes proper diagnosis and conservative judgment as to probable value. It has been the purpose of this paper to discuss some of the values and some of the limitations of this product.

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CHOLECYSTOGRAPHY: WITH SPECIAL REFERENCE TO THE DIAGNOSTIC VALUE OF THE EMPTYING TIME

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Cholecystography, since its introduction by Graham and his associates in 1924, has been the means of establishing many important facts relative to the anatomy and physiology of the biliary tract. Much of this knowledge has been utilized for the detection of pathological conditions of these parts based on variations from the normal as revealed by it. Today this test is considered an indispensable aid whenever disease of the gallbladder or its ducts is suspected. Most of the facts relating to its clinical application are so well known and generally accepted that only brief mention will be made of them. Response of the dye-filled gallbladder to a fat meal, routinely observed and recorded as part of the test by practically everyone using it, has been variously evaluated but the importance of delayed emptying as an index of disease is still a matter of controversy. It is the object of this paper to present pertinent observations of this phase of cholecystography as culled from the literature and personal observations.

The following cholecystographic findings are generally recognized as the most reliable basis for interpretation.

(1) Visualization of a homogeneous dense gallbladder shadow after an appropriate interval following administration of the dye and contraction of it after the fat meal signifies a normally functioning gallbladder.

(2) Nonvisualization of a definite gallbladder shadow indicates impairment of function to receive or concentrate bile and warrants a diagnosis of pathological changes.

(3) The visualized gallbladder can reveal nonopaque gallstones or benign tumors within its lumen not demonstrable otherwise. It can also identify opacities of doubtful origin as being within or outside of it.

The less reliable and more controversial findings revealed by cholecystography, serving as indices of disease, include:

(1) Faint visualization of the gallbladder.

(2) Persistent variations of shape or contour.

(3) Lack of satisfactory response to the fat meal.

It is not within the scope of this paper to give detailed information of the accuracy of the method as checked by operative procedure in connection with all of the findings listed. There is close agreement of statistical data compiled by numerous competent observers in regard to the more reliable signs indicative of pathological lesions approximating about 98 per cent. accuracy. Kirklin[1] of the Mayo Clinic in a recent report states that 95 per cent. of cholecystographic diagnoses whether positive or negative should be confirmable at operation. Of the less reliable findings according to this author faint visualization as an indication of impairment of function by disease should prove erroneous in less than seven per cent. if the designation "faint" be restricted to a shadow so delicate that it is hard to discern. Obviously the degree of diminution of shadow density suggesting pathology will vary with the personal equation of the observer and the quality of the films considered satisfactory, for which reason there is little agreement as to the reliability of this finding. Irregularity of contour of the visualized gallbladder is perhaps the most questionable finding of disease of the tract. Congenital bands, anomalies of development or pathologic conditions of adjacent structures, may be responsible for them and thorough investigations, including opaque meal studies of the gastro-intestinal tract, ought to be made before drawing conclusions as to its origin.

A few words about technique may be apropos in regard to accuracy of interpretation. At present the oral method of dye administration is generally accorded preference over the intravenous and if followed with meticulous care should prove equally reliable. Many variations of technique have been suggested for which special advantages are claimed by the users. All told, however, they appear to make little difference in the ultimate findings as reported. The double dose method does eliminate the need for reexamination in some cases which is of definite value. In a general way only technically satisfactory films should serve as a basis for interpre-

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tation and questionable findings ought to be checked for a confirmation.

Of all the findings revealed by cholecystography the impression that an apparently normally functioning gallbladder is in fact a normal one has been proved by operation to be the one most often in error. Estimates of the percentage of error vary widely but ten per cent. would appear to be a fair average. The facts that there is no definite normal standard of density or of contraction response to the fat meal, are largely responsible for this discrepancy. Faulty technique or interpretation may account for some but not all of those errors. It is in such apparently normal cases that an opaque meal examination combined with the dye test (as suggested by one of us)[30] may help to increase the accuracy of diagnosis. If it could be definitely determined that delayed emptying of such normal-appearing gallbladders is pathological, the percentage of errors in this group of cases could be further reduced. This study was made primarily to advance such a possibility.

Before considering the physiologic and possible pathologic aspects of contraction of the visualized gallbladder after a fat meal it may be well to review briefly some of the observations recorded in the literature as to the value placed on this finding by different workers. Kirklin[1] states that convincing proof is lacking that accelerated or retarded filling or emptying of the gallbladder as indicated by the succession of shadows is indicative of disease. McWhirter[2] and Hodges and Lampe[3] likewise attach little pathologic significance to films made after the fat meal other than the advantage which increased density and altered relationship incident to contraction may give to visualize stones or tumors which may not be seen otherwise. Sussman[4] in a recent article concludes that organic or functional pyloric obstruction to the passage of the fat meal is the most important factor accounting for variations in the emptying time of the gallbladder.

On the other hand, Cole,[5] Stewart and Illick,[6] and Golden[7] believe that delayed emptying of the gallbladder is indicative of disease. The latter two make one exception, namely delayed evacuation in patients with general atony. Watkins and Mills,[8] in an analysis of 625 cases examined by the oral method, interpreted 236 as showing functional disability as evidenced by delayed or poor emptying according to criteria which they had established. Fifty of these cases were operated on and in 48 cases evidence of a pathologic gallbladder was found. Newcomer, Newcomer and Conyers[9] in an anlysis of 360 cases, examined by the intravenous method found 50 in whom they interpreted delayed emptying of the gallbladder which they regarded as diagnostic of chronic cholecystitis. Thirteen of these were operated on and 12 of them were found to have pathological gallbladders. Of 75 cases examined by them by the oral method fifteen showed similar findings and four of these when operated on were found to have chronic cholecystitis. Casellas[10] in a recent report states that when a gallbladder retains over 50 per cent. of its contents three hours after the fat meal, no matter how good the concentrating power is, poor gallbladder function and potential pathology is present. He cites six cases showing delayed evacuation after the fat meal which, upon being reexamined five to thirty-eight months later still showed delayed evacuation and in addition calculi which had not been noted at the time of the first examination.

Rose[11] in 1934 in a review of 1,616 cases studied by cholecystography found 25 which had visualized well, but emptied slowly after the fat meal. These were operated on. Twenty-four had chronic cholecystitis, 12 with stones; one case had slight cholecystitis but no stones. Feldman[12] in 1930, in a series of 500 cases found 46 with what he considered abnormal contraction after the fat meal, but gives no specific operative findings for this group.

Chandler and Newell[13] also cite five cases which at operation showed gallbladder disease, in whom delayed emptying after the fat meal was a striking feature.

The following facts relating to the function of the normal gallbladder are generally recognized. Bile which has collected in it and been concentrated by its mucosa is expelled by tonic contractions of its muscular wall through the cystic and common ducts into the duodenum. Contractions are initiated by the hormone cholecystokinin which is derived from the duodenal and upper jejunal mucosa in response to fat digestion. Simultaneously with the contraction of the gallbladder the sphincter of Oddi and the duodenal musculature relax, thus permitting entry of the bile into the duodenum. There is probably a reciprocal functional relationship

between the gallbladder, the duodenum and the sphincter of Oddi to produce this type of action. Nervous reflex mechanisms and other factors are thought to play a role the exact importance of which is not as yet clearly established.

According to Boyden[14] who has made extensive studies of gallbladder emptying with cholesystographic aid, evacuation after an egg yolk-fat meal occurs in successive phases of contraction. Following a latent period of about one minute after ingestion of the meal a small amount of bile is discharged during the first two minutes. A short pause then occurs after which the principal period of discharge takes place. From 50 to 75 per cent. of the contents are evacuated in from 16 to 60 minutes (average 32 minutes). A period of quiesence then follows lasting five to 45 minutes after which another phase of contraction occurs which results in the emptying of a variable amount of the residue. One or more additional phases usually lead to almost complete emptying, the time for which varies markedly in different individuals. Similar observations have been made by others and a decrease in volume of at least 50 per cent. one hour after the fat meal, which is the common interval chosen for examination, is generally regarded as a normal response.

In view of the known facts of normal emptying, failure to do so may then be ascribed to the following factors:

1. Absence of cholecystokinin formation.
2. Dysfunction of the gallbladder musculature.
3. Lack of patency of the cystic and common ducts.
4. Improper function of the normal reciprocal mechanism of the gallbladder, duodenum and sphincter of Oddi.
5. Reflex disturbances.

The problem of cholecystokinin production and consequent gallbladder contraction in connection with cholecystography probably bears a direct relation to the fat meal passing through the pylorus and undergoing some digestion. According to Ivy[15] good fat digestion should speed the rate of evacuation. If pyloric obstruction be present hormone formation may be delayed and evacuation be interfered with. Sussman[4] cites two cases which would seem to confirm this assumption. Boyden and Ritchie,[16] however, in a study of seven cases of carcinoma of the stomach with some degree of pyloric obstruction as observed by the opaque meal examination, found that normal gallbladder visualization and contraction occurred in all of them. These variations can probably be explained on a quantitative basis, i. e., if enough of the dye and fat meal pass through the pylorus a normal response occurs, otherwise not. Sandblom[17] on the basis of a study on gallbladder evacuation after stomach surgery, states that in such cases delayed evacuation, observed shortly after operation, disappears as stomach motility improves.

Providing cholecystokinin has been formed in sufficient quantity the next essential for a normal emptying mechanism is the presence of a gallbladder musculature, able to respond to it. Experimentally Murphy[18] has produced acute cholecystitis in dogs by the intravenous injection of eusol. The resulting lesion involves especially the fibromuscular layer and also extends into the elastic and subepithelial layer. The mucosa is usually not involved. Both by direct observations and by roentgen studies it was found that these gallbladders did not empty after a fat meal. In only one instance in which the cholecystitis produced was mild did the gallbladder empty, but then only to a small degree. Casellas[10] is of the opinion that delayed emptying in many cases is due to a similar lesion in the human, that is a pathological alteration of the muscularis which prevents its normal function. The mucosa, lymphatics and blood vessels are sufficiently uninvolved so that the gallbladder visualizes normally.

Patency of the biliary ducts of necessity plays an important role both in visualization and evacuation of the dye-filled gallbladder. Complete obstruction of the cystic duct will obviously prevent visualization and similarly complete obstruction of the common duct of any duration will cause an obstructive jaundice with resultant failure of visualization. Partial obstruction of either duct may allow normal visualization but may result in delayed evacuation. Cole and Rossiter[19] have listed the following causes for partial or temporarily complete obstructions in the cystic duct:

1. Stenosis produced by surrounding adhesions.
2. Stenosis produced by a thickened wall.
   (a) Due to acute inflammation.
   (b) Due to diffuse fibrosis.

3. Congenital or inflammatory twists or kinks.

4. Congenital or inflammatory lesions involving the valves of Heister or fibrous strands.

5. Stone in duct.

6. Tension induced by enlarged liver.

7. Compression or filling defect due to tumor or lymph nodes.

8. Obstruction due to anomalous hepatic or cystic artery.

Common duct lesions will be given consideration in connection with abnormal reciprocal mechanisms about to be described.

A discussion of the disturbances of the gallbladder, duodenal and sphincter of Oddi mechanism and its relationship to delayed emptying of the gallbladder may be divided according to the following groups:

1. The functional group, or so-called dyskinesia, of which according to Eestphal, et al[20] there are two types.

(a) The hyperkinetic hypertonic type in which there is contraction of the gallbladder with spasm of the sphincter of Oddi.

(b) The atonic type, with relaxation or atony of the gallbladder with spasm of the sphincter causing atonic distension of the gallbladder.

2. The organic group comprising lesions of the common duct, duodenum and sphincter of Oddi.

There have been many clinical and experimental observations concerning the functional type of sphincter of Oddi obstruction and its relationship to biliary stasis and gallbladder symptomatology. The accepted criteria of a diagnosis of biliary dyskinesia as given by Carter and Hotz[21] are: (1) An irregular symptom complex of pain in the R.U.Q., or colic, nausea, distention, diffuse tenderness and vomiting; (2) A cholecystogram showing normal concentration, with delayed evacuation; and, (3) biliary drainage showing a failure of the gallbladder to respond normally to stimulation with magnesium sulphate and olive oil.

Ivy[22] reports the following interesting observations in the human. A duodenal tube was passed and bile obtained. This was followed by a cessation of bile flow probably due to contraction of the sphincter of Oddi. No distress was experienced at this time. Then a secretin-cholecystokinin solution was injected intravenously. This usually causes a copious flow of bile and pancreatic juice. In this case, however, only pancreatic juice was obtained and ten minutes later hypochondrial distress was experienced which gradually became more severe. Magnesium sulphate was then introduced into the tube. This resulted in relief of the distress after a few minutes, and after seven minutes dark bile was obtained. This observation confirms the hypothesis of Metzler, as reported by Ivy,[22] that magnesium sulphate placed in the duodenum may relax an abnormally contracted sphincter of Oddi and also that a normal response of the gallbladder to cholecystokinin in the face of a spastic sphincter of Oddi results in clinical symptoms of gallbladder disease. Westphal[20] performed a similar experiment in which pilocarpine was used. This causes gallbladder evacuation but contraction of the sphincter of Oddi. Following the injection of pilocarpine, the passage of bile into the duodenum stops and biliary distress occurs. The symptoms are relieved by atropine which abolishes the action of pilocarpine on the gallbladder and sphincter. Best and Hicken[23] have observed spasm of the sphincter of Oddi postoperatively, by cholangiography, in cases with cholecystitis and cholelithiasis.

Boyden and Gerdes[24] believe that retardation of emptying of the gallbladder in the second and third trimester of pregnancy is probably due to spasm of the sphincter of Oddi, a normal physiological dyskinesia reflecting the changed human content of the organism in pregnancy.

An increase in the muscular tone of the duodenum may inhibit the flow of bile into the duodenum. Crain and Walsh[25] have experimentally produced a duodenitis which caused a delay in evacuation of the gallbladder. Strauss et al[26] reported a series of cases with chronic biliary stasis, which they interpreted as being due to an inflammatory process of the lower portion of the common duct with thickening and narrowing of the ampulla of Vater. These conclusions were based on surgical findings. The inflammation of the gallbladder in these instances was mild. Experimentally in dogs, Shapiro and Kasabach[27] produced duodenal ileus. This resulted in an increase in the evacuation time of the gallbladder. Orndoff[28] is of the opinion that one of the causes of biliary stasis in pregnancy is the duodenal ileus which is often seen in that condition.

Although the role that reflex mechanisms play in gallbladder evacuation are not definitely known, certain observations have been made

which indicate that such mechanisms may have some influence. Carter and Hotz[21] recently reported three cases in detail with classical signs of biliary dyskinesia including normally visualizing gallbladder with delayed emptying in whom appendectomy was performed because of associated signs and symptoms suggestive of appendiceal disease. Medical treatment had been of no avail. All three were relieved by appendectomy which also resulted in a return to normal of the emptying of the gallbladder on cholecystography, and in return to normal findings with duodenal drainage. In all three cases, the gallbladder was grossly normal in appearance at the time of surgery. These authors believe that these were cases of spasm of the sphincter of Oddi of reflex origin, due to disease of the appendix. They cite several European investigators who have had similar experiences. Experimentally in dogs Oughterson and Mendillo[29] found delayed evacuation of the gallbladder following the removal of the appendix, mechanical irritation of the terminal ileum by rubbing with gauze, cautery incision of the appendix and in some instances by just opening the abdomen. This was associated with delayed emptying of the stomach but they believe that "even though the emptying time of the stomach is delayed there is a frequent passage through the pylorus of small amounts of stomach contents sufficient to stimulate the duodenum." They suggest that dysfunction and stasis of the gallbladder may be due in part to inhibitory reflexes arising from chronically diseased portions of the gastro-intestinal tract.

Our study of cases showing slow emptying of the visualized gallbladder after the fat meal was begun about 16 months ago. It was initiated at the instigation of Dr. Warren H. Cole, who felt that conclusive observations relative to this phase of cholecystography were lacking and additional information was desirable. As a preliminary check or control, films of 50 cases previously examined and classified as normal were reviewed with a view towards estimating the amount of decrease present on the film made one hour after the fat meal. It was found that the shadow had decreased anywhere from 50 to almost 100 per cent. on them with an average of about 75 per cent. It was decided arbitrarily to consider all cases showing more than 50 per cent retention after one hour as suitable for further investigation.

Since then, of 655 cholecystographic examinations made, 41 were originally selected as possibly conforming to our standards of delayed evacuation. A subsequent more critical review of these led us to eliminate eight as being questionable leaving 33 or about five per cent. as showing definite slow emptying. The amount of retention varied all the way up to 100 per cent. All but four of them showed good concentration and the degree of diminution of density of these four was hardly sufficient to designate it as faint. Three showed persistent deformity and four nonopaque stones. In one there was a complicating pyloric obstruction, another had a questionable gastric ulcer and a third developed intestinal obstruction due to carcinoma of the sigmoid shortly after the gallbladder examination.

Ten of the patients were operated on and nine of them were found to have diseased gallbladders. Eight had gallstones but in only four of these were they observed on the original films. (Reexamination of the films after the operation again failed to reveal any shadows which might have been interpreted as stones.) The one case which showed a normal gallbladder at operation showed extensive adhesions involving the biliary ducts. One other case not included in the operative group because no attempt was made to investigate her gallbladder (she was operated on for colon obstruction) was of particular interest. She had a dye test ten days before she developed signs of intestinal obstruction at which time nonvisualization was noted. The plain film made ten days later when a tube decompression was being done showed a well distended dense gallbladder shadow, although she had been on a restricted diet during the interval. In her case there apparently was delayed filling as well as delayed emptying.

Of the cases which did not come to operation, although they all had typical gallbladder histories 13 had check-up dye examinations at various intervals after the original one. In eight of these the findings were practically identical on the two examinations. Three showed a normal fat meal response on the second examination, one had nonvisualization after an interval of five weeks and one showed a diminished density after three weeks. In several of the check-up cases with delayed evacuation successive films were made up to five hours after the fat meals all of which showed about the same degree of reten-

tion. Two were given a second fat meal two hours after the first one and succeeding films again failed to show normal emptying.

From some of the information obtained from the literature reviewed and personal observation it would seem that delayed emptying of the gallbladder after the fat meal as an index of disease deserves greater consideration than it has received. There is adequate evidence that bile stasis eventually leads to inflammatory changes or stone formation and earlier recognition of it with adequate treatment may obviate some of the after effects. Patients showing delayed emptying cholecystographically certainly ought not to be regarded as having normal function, but their gallbladders should be considered actively or potentially pathological until proved otherwise. Such a finding ought to stimulate efforts to determine a possible cause for it and remove it if possible.

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THE PROGRESSIVE IMPORTANCE OF THE ROLE OF THE PRACTICING PHYSICIAN IN TUBERCULOSIS CONTROL*

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INTRODUCTION

Because of the protracted form of illness, heavy mortality and many other grave features, tuberculosis has always been the physician's responsibility, but only recently has this responsibility been adequately realized and duly accepted by the medical profession. For centuries an attitude of mingled ignorance, indifference and fear hampered medical progress. As a result, hu-

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mane lay people had little choice but to take over tuberculosis as a ward of the community. Since Villemin and Koch, however, there has been a a gradual transition from the *passive* attitude in handling tuberculosis to positive and *active* treatment of the disease.

A gradual unfolding of the knowledge of tuberculosis will be sketched to show the reasons for the changing attitude. In addition, the recent gain in sentiment to profit from this knowledge will be pointed out. Finally, some of the most recent aids in diagnosis will be presented to stimulate the profession to further progress.

Progress was slow at first because the century old superstitions and phobias about tuberculosis did not disappear quickly even after the true cause of the disease was known. Although Brehmer and Dettweiler's rest treatment was applied by Trudeau, and the teachings of Koch and Villemin by Biggs and others, the disease was too deeply rooted into the race to show striking results at first or to change opinions rapidly.

EARLY FUNDAMENTAL WORK

Advancement seemed to await the "magic key" of knowledge, which was supplied to a great extent by medical research.

Beginning with Parrot in 1876 on through the work of Conheim, Cornet, Küss, H. Albrecht and E. Albrecht, and more recently Ghon, Ranke, Opie and others, there was revealed the secrets of primary tuberculous infection. The calcified foci or "tombstones" found in so many people by Naegeli, Orth and others, began to take on significance. The pathogenesis of the disease was being better understood. The new conceptions of the disease course taken with the discovery of the tubercle bacillus seemed to set the stage for a rebirth of activity which began to show results about twenty years ago.

The x-ray, discovered in 1896 by Roentgen, came into use in other branches of medicine at the turn of the century, and has recently taken first place in the diagnosis of tuberculosis. The x-ray shadows revealed for the first time the tuberculous lesions *during life*. It was something new and tangible. As time went on extensive lesions were discovered that couldn't be heard by the most skillful clinicians. At the present time the earliest lesions may be seen many years before they produce illness or clinical findings. In addition, lung cancer, abscess, bronchiectasis and many other diseases may be more readily separated from tuberculosis. Thus the roentgenologists joined with the increasing numbers of physicians, nurses and socially minded people to help the tuberculous.

Pneumothorax, which was used little immediately after Forlanini's work in 1882, has been found to be a therapeutic agent of the first importance, and during the last two decades has been applied to about half of all tuberculous patients. Paralleling pneumothorax, Brauer, Sauerbruck, and later many other surgeons, found that surgery would not only shorten the convalescence of certain severe cases, but would cure them entirely.

Tuberculin, prepared by Koch as a therapeutic agent, and which proved to be a failure as such, was found by Pirquet and Mantoux to be a diagnostic aid in children. More recently it has been adopted as a universal "tool" in the control of the disease.

THE DISEASE PATHOGENESIS

A pacemaker of these many advances, as alluded to before, was a better understanding of the epidemiology, infection, immunology, pathology and pathogenesis of the disease. A composite sketch of these phases of the disease will be drawn from a more elaborate abstract of the work of many authors reported before,[1] and this knowledge will be shown, not only as a pacemaker but actually the foundation of medical understanding and progress in tuberculosis.

After the tubercle bacillus was discovered, tuberculosis was soon found to be a generalized or systemic infection which only gradually becomes focalized in various organs as healing takes place. During the "generalization" phase there may be an ephemeral temperature or "initial fever"[2] lasting from two to ten days, after which few symptoms occur until advanced disease appears. The interval between the two clinical episodes has an average of about ten years.

The infection may take place at any time during life and may be repeated many times. Generally "reinfection" on patients having active lesions only occurs when the dosage of bacilli is large and all healed foci give some protection against new infections. The majority of primary infections (about 80 per cent. in America) take place in the lungs; about eight per cent. in the bowels; and about two per cent. in the head region including the tonsils, and the other ten per cent. in other rare portals of entry or various

combinations of the ones mentioned. The rate of infection varies widely in different countries, even in different communities, but is generally decreasing. This decrease tends to throw more primary infections into adult life. The average infection rate today is between one and two per cent. per year, contrasted with five to ten per cent. fifty years ago.

The first, or primary, infection in children occurs as varying sized patches of bronchopneumonia in the lung. From the local focus the bacilli follow the lymphatic vessels and form lesions in the various lymph nodes along the course of the lymphatics. The infection finally reaches the left subclavical vein whence it travels to the right side of the heart and becomes disseminated in the lungs. The bacilli many times pass through the pulmonary capillaries and return to the left side of the heart, and from there to the general circulation to become deposited in all the organs of the body. Occasionally these foci develop into extrapulmonary disease.

The lesions are located anywhere in the lung parenchyma with a slight predominance in the bases. The majority of the lesions heal during the first seven to eight years and after that time may become completely resorbed. All but an insignificant number of infections due to small dosage heal, but only about 75 per cent. of patients heal where there has been heavy exposure. The lesions that do not heal may spread slowly by a growth or rupture in the bronchi or blood and lymph vessels. The infection may also spread from the lymph node lesions, which may overflow and rupture. The bacilli may enter the blood stream, get into the general circulation, and cause a systemic dissemination. As mentioned above, the average "latent" or "quiescent" period from the time of infection to the appearance of clinical disease has been found to be about ten years.

Primary tubercles of adults[3] on the other hand usually involve the lymph nodes less, have poorer capsules, and tend to localize more in the upper lung lobes. This results in a smaller number of protracted primary "sequelar" lesions and exacerbations in the adults compared to those found in the lymph nodes of children. For the same reason there is prone to be a more rapidly progressive pulmonary process in the adults. In brief, adult primary type produces disease it proceeds rapidly and is confined largely to the lung parenchyma. The average "latent period" is usually about three years. The exact number of adult primary lesions that heal is not known, but it is perhaps equal to or greater than those found in children.

After the first infection, there is an acquisition of hypersensitiveness of the body cells to tuberculin, called *allergy* by Pirquet. This "sensitizaton" of the body cells is shown by the tuberculin skin reaction appearing from three to nine weeks after infection. A relative immunity also develops largely independent of the allergy,[4] although the latter in moderate degree may contribute to the resistance to infection. In excessive degrees allergy may aggravate the infection.[5, 6] The effects of these changes in the basic nature of the host tend to cause a localization of the process in the body organs and tissues, most of which occur in the lungs.[7] This "organ localization" only develops gradually and is never complete except in cases that are healing. Even healing lesions may become "generalized" as a miliary tuberculosis under certain accidental provocations.

The disease may continue as a "progressive primary lesion" or an "exacerbation of a primary lesion." An example of the latter type will be illustrated by a case history with rotentgenograms and photographs.

Case 1: B. H.: (A1046) was a girl 21.4 years old at the time of death. There was no known contact with tuberculosis and very little in her early history was pertinent to this discussion. She worked as a package opener in a department store from April to September, 1936. In July she felt "pepless" but in September she coughed and spat blood. A diagnosis of tuberculosis was made on x-ray, and tubercle bacilli were found in the sputum. The disease seemed to be localized in the left upper lobe, with cavity formation. A pneumothorax was given which collapsed the upper lobe but not the lower. All seemed to go well for a time, but after admission to the sanitarium the patient suddenly began to run a high temperature, developed fluid in the right base, and went down rapidly to death six months later.

At the autopsy in May, 1937, there was found an old calcified primary lesion in the left base, made up of several different foci, with a corresponding lymph node complex up the bronchi and at the hilum. One focus had apparently overflowed and produced a large nodular mass that ruptured into a bronchus. No doubt this was the source of many subsequent reinfections. There was a cluster of more recent calcifications involving an area supplied by the left subapical bronchus. This was a reinfection, probably originating from the open lesion in the base, and was of decidedly more recent age

than the primary. A small dilated terminal bronchus may have represented a healed ulcer, but no other signs of the cavity were found. There were various types of dry caseous lesions in the left base and left upper lobe. Some were slightly calcified. The ulceration of one of these foci into a small cavity perhaps represented the onset of clinical disease with hemorrhage one year before death. The type of recent lesion throughout the left base seemed compatible with one caused by the pulmonary hemorrhage, because there were acinous nodose masses, some rather large, surrounded by hemorrhagic lung tissue. An endobronchitis was present in some bronchi. There was a recent spread throughout the right lung with basilar pleuritis, and evidence of fluid was present. There was still considerable atelectasis in this base and some masses of tuberculous infiltration. The right upper was encased in an adhesive tuberculous pleuritis, and in the lung parenchyma were scattered acinous nodose clusters about every 7 mm. The middle lobe was similar.

Comment: This individual was probably infected from ten to twelve years before death from an unknown source. The primary disease developed in the left lower lobe with a central primary lesion and two or three satellite tubercles. It spread up the lymphatics to the hilum leaving behind old calcified tubercles. The local lesion in the parenchyma "overflowed" and opened into a bronchus some time afterwards and spread the infection into the upper part of the lobe, forming the subapical lesion of the left upper lobe. These lesions smouldered on as daughter-type colonies, one of which opened up into the cavity that was perhaps described by the doctor's first report and which no doubt formed near the time of her first hemorrhage. After admission into the sanitarium the pneumothorax closed these cavities and probably assisted in healing them as well as the foci in the left upper lobe, but because the open cavity in the lower lobe was not seen on the x-ray a complete pneumothorax was not deemed necessary. This lesion doubtless caused a spread across to the right upper and right lower lobes, resulting in an acinous nodose spread by the bronchi and a miliary spread by the blood or lymph vessels that caused the terminal miliary tuberculosis. The actual entry into the blood or lymph streams was not found.

This case is reported in detail because it reveals in bold relief one of the common means of transition from first infection to tuberculous disease.

The disease may also result from a much less frequent "exogenous reinfection from without." When these "reinfections" locate in the lung they do not differ much roentgenologically in size or position from the adult primary lesions with which they may be easily confused.

The majority of "reinfection" lesions are found along the subapical bronchi, while the next in frequency, but slightly more benign in character, are those located in the apexes. Next in frequency are the apexes of the lower lobes and finally the horizontal rami of the upper lobes become involved. In brief, the posterior upper quarter of both lungs are the sites of predilection in over 90 per cent. of the early cases.[8]

On the roentgenogram the lesions appear as small circumscribed or cloudy flecks a few millimeters in diameter.[9,10] These lesions may form "reinfects" or daughter colonies which may proceed to ulceration and then scatter the infection over the lungs by the way of the bronchi, finally resulting in fibrocaseous and ulcerative disease. The disease usually spreads forward and downward from the smaller early lesions located in the posterior upper quadrant.

The infection may spread to the other organs by the blood or lymphatic routes as it usually does during or after the primary infection. Lesions thus form in the kidney, liver, spleen, adrenal, meninges, skin, bones and many serous cavities. Direct implantations of bacilli cause laryngitis, enteritis and cystitis. The whole gamut of changes are found, from miliary tubercles to caseous pneumonia. Every specialty of medicine may thus be involved and no practitioner should consider himself prepared without a basic knowledge of the various tuberculous complications.

Following in the wake of the disease, nonspecific "sequelar" lesions occur. These may be conveniently divided into the toxic, such as amyloidosis and fatty changes; obliterative, such as those in the adrenals leading to Addison's syndrome; and those resulting from fibrous contraction. The latter includes the distressing tracheobronchitis; bronchiectasis; bullous emphysema, and finally heart failure due to fibrous obliteration of the pulmonary capillaries and arterioles.

There are also rare lesions thought to be caused by atypical tubercle bacilli which cause diagnostic difficulties. The disseminated follicular lupus, Boeck's sarcoid and perhaps such conditions as Poncet's rheumatism and Pick's cirrhosis may be included in this category.

Apart from the preceding conditions caused directly or indirectly by the tubercle bacillus there is the whole gamut of diseases that may be associated with tuberculosis as a concomitant or associated process. Each one requires skill in separating from tuberculosis. Diabetes, syphilis, cancer, heart disease, nephritis and especially silicosis and silico-tuberculosis are important in this group. Further than this the many compli-

cations of tuberculosis may be confused with non-tuberculous complications.

It can thus be seen how this many-sided disease can only be a doctor's problem, and that the accumulated knowledge has given the physician a tremendous advantage in the diagnosis and treatment of tuberculosis. This progress also carries with it an obligation for the physician. Just as the tuberculosis physician took the stage from the lay worker, in like manner there the hands of the general practitioner as he will absorb. Clinics, lectures and post graduate instruction are the principal courses followed. The Sanitarium and Dispensary staffs also act as free consultants in handling or giving advice in the treatment of tuberculosis. Similarly with Plunkett, Miller and Amberson, in New York; Draper, in Indiana; Bellinger, in Oregon, to cite only a few isolated instances over the country, this system has prevailed. The system has been found

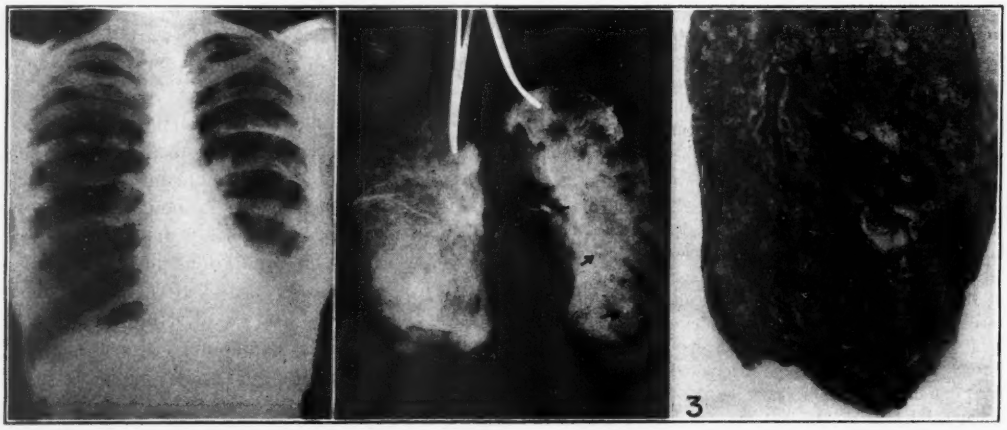

Fig. 1. Antemortem x-ray taken on November 12, 1936, showing a slight effusion, slight atelectasis, and nodular lesions in the base of the left lung; a pneumothorax in the left upper lung with nodular calcifications in the lung parenchyma.

Fig. 2. Post mortem x-ray of the lung of the same case. Note the irregular calcifications in the left upper, the confluent acinous nodose process in the left lower part of the lung, and the rather diffuse broncho-pneumonic spread in the right lung. The most important feature is the primary lesion in the center of the left base with lymph node components up the bronchus.

Fig. 3. A sagittal section of the base of the left lung through a large fibrocaseous mass in which are found several dense calcified foci corresponding in character to the primary lesion. *This nodular mass has communicated with the main bronchus.*

is a wide open opportunity for the general practitioner to supplant the tuberculosis specialist in case finding.

RECENT TENDENCIES IN TUBERCULOSIS CONTROL

The recent tendency in tuberculosis control is recognizing the opportunities presented and taking advantage of them. It is recognized that the medical man in the home has the best opportunity and is in the majority of cases the only one to see the disease early.

In Detroit, Douglas[11] has observed that about 75 per cent. of their diagnoses of tuberculosis are made by the general practitioners. So important do they consider the doctors that the Health Department has taken the whole profession in as special deputy health officers to share in tuberculin testing and other activities. Tice and Hruby[12] in Chicago have put as much work in to be effective because *it takes the men into the work who first encounter the patients having tuberculosis.*

Recently Pleyte, Holand and Phillips[13] have reported on surveys by the Wisconsin Anti-Tuberculosis Association in the State of Wisconsin that leave little doubt about the role the general practitioners can and should play in case finding. Utilizing the old Framington ratio of "nine times the number of active cases per death from tuberculosis" Pleyte estimated that Wisconsin, with an annual average of 984 tuberculosis deaths for 1936-38, has 8,856 active cases or one active case for every 342 persons in the general population. With this "normal expectancy" of one case per 342 unselected population as a base, comparison of x-ray case finding productivity was made for various classifications of x-ray

patients. In 2,905 "youths" (under age 20) who were tuberculin reactors or contacts, the rate of findings on x-ray for "suspiciously active reinfection tuberculosis" was 2.8 per 342 studied, or 2.8 times the estimated normal expectancy for the average Wisconsin population. In tuberculin-positive W. P. A. workers the rate was 7.7 times the normal expectation. In this group were many Negroes. Among tuberculin-positive inmates of the Milwaukee County House of Correction the rate was 10.2 times normal expectancy.

In 1,377 adult cases, 88.5 per cent. of whom were selected for x-ray because of contact, positive tuberculin reaction or previous tuberculosis diagnosis, the rate of findings was 17.9 times normal. The tuberculin-positive cases (but without known contact), forming 31.8 per cent. of this adult group, showed a rate 6.2 times the normal rate; the "contact" group, which made up 46.0 per cent. of the adult group, had a rate 13.5 times normal; and the "previously diagnosed," comprising 10.7 per cent. of the group, had 79.1 times the expected normal rate of findings. For two county asylums studied, the rate was 20.2 times the normal, confirming the high findings of Hilleboe, Burns, and others.[14]

The most important classification from the angle of case finding productivity was a group of 2,159 films sent by private physicians to the Wisconsin Anti-Tuberculosis Association for consultation. These films are obviously of "suspect cases"—of patients having had contact to an open case, who were ill, or had been or were suspected of being ill. Among these films the rate of suspiciously active findings was 40 times the estimated average morbidity rate for the state. Dr. Pleyte reveals a tremendous growth of this consultation service due largely, he thinks, to the results the doctors are getting from it. This work clearly demonstrates that early diagnosis lies principally in the field of roentgenology and that *the general practitioners must take the x-rays or order them taken.*

ADDITIONAL AIDS FOR THE PHYSICIAN: TUBERCULIN

In addition to a "consultation service" on x-rays, or to the advantages of an x-ray survey, there is also valuable information obtained by intelligent tuberculin testing, which might be called the twin of the x-ray so far as diagnosis is concerned. Tuberculin testing has become a diagnostic agent of the first magnitude with error less than one per cent. It is suprising to find out how many negative reactions are found in lung cancer, abscess, bronchiectasis, Hodgkins' disease, sarcoid, coccidioidal infection, etc., where there may be a question of tuberculosis. Furthermore, many people today reach adult life without infection. A strongly positive reaction, although not diagnostic, is suggestive of an active lesion. In young children it is presumptively diagnostic.

During the last fifteen years there has been a widespread use of tuberculin testing in school children. This procedure has been of most value in rendering people "public health conscious," although it is also a useful case-finding device as well. It seems to be the feeling at present, however, that wholesale tuberculin testing in schools is not getting as good results as selective testing of groups, especially contacts, as Pleyte and others have done.

Tuberculin is also of great value in the doctor's office. Every physician should be able to dilute old tuberculin or dissolve P. P. D. and inject it under the epidermis, as Mantoux originally recommended. Those who feel unable to do it should go to the local tuberculosis physician and learn the technic. The same should apply to roentgenographic technic and interpretation. If the tuberculin test is positive it should generally be followed by a roentgenogram.

The procedure for the study of patients is not a difficult task. If there is a history of tuberculosis in the family the physician should tuberculin test all those who have been in contact with the open case. He should then x-ray or fluoroscope the tuberculin-positive cases at regular intervals to catch any developing disease which appears in about one in four and which kills about one in forty heavy exposures. Any suspicious symptoms such as chronic cough, loss of weight, loss of appetite should be similarly studied. An ideal situation will be the time when routine tuberculin testing and x-raying will be done on all patients coming to the doctor's office or to a hospital.

ELIMINATING CONTAGION

There cannot be too much emphasis placed upon the danger of the open case. Recently in Illinois at the Manteno Insane Asylum there

was much criticism of officials for the deaths of 52 insane patients from typhoid fever, but people are rather indifferent to the fact that about 3,000 or more die every year in the state from tuberculosis. Yet the responsibility is virtually the same. Both disease are infectious and deadly, but tuberculosis kills only after a decade or more while typhoid kills more quickly. There is one other important difference. Typhoid may be obtained from water, food or contaminated objects, while tuberculosis is much more a disease of direct contact to an open case.

REFINED LABORATORY METHODS

The advantages afforded by other aids may be brought into use for the cases having lung symptoms. Systematic culturing of sputums and other body fluids may be ordered which may not only discover tubercle bacilli but may also reveal other pathogenic microorganisms. The streptococcus, the staphylococcus, the Friedländer bacillus, B. pyocyaneous, and other causes of abscess may be thus discovered. Anaerobic cultures frequently yield interesting findings, most important of which is the Welch bacillus. In addition to the usual pathological sections, bronchoscopic specimens may yield valuable clues regarding cancer, tuberculosis, or benign tumors. Our own experiences will be constructive in this connection.

In the admission ward of the Municipal Tuberculosis Sanitarium of Chicago during the last four years and eight months, 2.79 per cent. of 6,304 cases were found to be other conditions than pulmonary tuberculosis, although they simulated tuberculosis (table I). About five per cent. more had another clinically concomitant disease with tuberculosis. In the autopsy service 7.5 per cent. of the autopsies were found to have no tuberculosis at all. Furthermore, there were few autopsies on tuberculous patients that did not show some other complication than tuberculosis.

The high percentages of diseases other than tuberculosis, shown by our autopsy findings, however, are misleading because they always reflect the zeal of the medical staff in obtaining autopsies in unusual cases. For instance, the lung cancer figures for our autopsy series during seventeen years are 28 on 1,252 autopsies (2.24 per cent.), while the percentage for admissions was only 0.17 per cent., about 7.5 per cent. of the autopsy figures. The lung cancers found recently on admissions during the four years and eight months are only 16 in 6,304 patients (0.26 per cent.)—one-ninth, or about 11 per cent. of the number. These figures may be made to harmonize with autopsy figures if completely analyzed. Inasmuch as our autopsy percentages over the years is about 33 per cent. of the deaths, and the total deaths are only about 30 per cent. of the admissions, the ratio of cancers to admissions would be only about ten per cent. of the ratio of the cancers to the number of autopsies. Allowing for the ones that were missed ten or more years ago, before diagnostic ability was as good as at present, the figures are quite comparable.

Fairly accurate figures, therefore, may be established on total admissions when careful studies are made in diagnosis, but not on autopsy findings. In recent years few patients leave without an absolute diagnosis either by autopsy, bronchoscopy, thoracoscopy, sputum section or lung puncture after death. Absolute figures can only be established on total population with absolute diagnosis on all deaths—a thing impossible at the present time.

Practically all other disease conditions can be analyzed in the manner just given for lung cancers. The lung abscesses were only about five times as high in the autopsy service. There were 0.72 per cent. in the autopsy series and 0.17 per cent. in the clinical series. Inasmuch as over half of these cases survived it would naturally increase the number in the clinical series compared to the autopsy series. For bronchiectasis there was a percentage of 0.64 in the autopsy series and 0.40 in the clinic. There were still more survivals of patients having bronchiectasis than those having lung abscess. In lues and bronchial asthma there were no uncomplicated cases at all coming to autopsy, so the clinical figures are the only ones that show anything.

The table reveals other interesting data useful to the physician. First of all, it shows that a tuberculosis hospital of our type (which is a fair representative of the larger institutions) admits 85.09 per cent. of patients that are positive on the first few days of sputum study by direct smear or by direct smear and concentration examination. According to previous studies the direct smear only reveals about 70-75 per cent.

positives. Ten to fifteen per cent. more are, therefore, gained by concentrating the sputum. Without a concentration, errors may result in 10 to 15 per cent. of cases. By examining the stomach washing or by repeated culturing the sputum, another 7.96 per cent. are added to the positive sputum cases. There were 4.16 per cent. of the cases that have definite x-ray evidence of a minimal tuberculosis or have had other evidence of having had tuberculosis before, but whose sputums were always negative while under our observation. This group is the most difficult of all on which to establish a diagnosis. Finally, there were the 2.79 per cent. of cases mentioned above having no pulmonary pathology of a tuberculosis nature excepting "healed calcified foci." Of this group 52 (or 0.80 per cent.) were cases who had an ephemeral pulmonary involvement which had cleared completely by the time the patient reached the admission ward (from one to several weeks later). There were 37 similar cases (0.57 per cent.) which showed only "healed calcified foci." There were eight cases of extrapulmonary tuberculosis which had nothing referable to the lungs.

Of the definite non-tuberculous diseases, bronchiectasis came first with 0.40 per cent. Only a few of these died and still less of them came to autopsy. The incidence of other diseases may be seen in the table. It is interesting to note, however, that a large variety of diseases may simulate tuberculosis so closely that they are sent in as tuberculosis. For example, *chronic passive congestion* as a result of a mitral stenosis is encountered. *Endocarditis lenta* is occasionally confused with tuberculosis. These mistakes, however, are not nearly as frequent as mistaking early tuberculosis for a vast number of other conditions some of which have been listed in the table. Naturally these mistakes vary with the skill and experience of the physician.

It may be seen from these observations that in spite of modern methods of diagnosis it is not always easy to find tubercle bacilli in certain cases of tuberculosis; that some cases do not show them at all; that a significant percentage of our cases are not tuberculous at all; and that even a higher percentage have concomitant complications or diseases. To make a diagnosis of early tuberculosis or to separate tuberculosis from other diseases requires careful observation and study even *in an institution where all the facilities for study are available.* How much more difficult is the problem for the general practitioner who has at the beginning only clinical facilities for study in a group wherein the percentage of tuberculosis compared to other diseases is much lower!

Since tuberculosis runs a course in years and decades, the patients in the quiescent or latent stages of the disease are all subject to practically every other disease. *The greatest problem of all, therefore, seems to be the identification of tuberculosis in the presence of other diseases, not only to detect tuberculous disease but to weigh the hazards of the complicating disease in activating the tuberculosis.*

The problem of a physician is therefore not only dependent upon his ability to detect the many diseases that confront him other than tuberculosis but also upon his knowledge of tuberculosis itself so that he may recognize it in its early stages or as it may be associated with other diseases and complications.

SUMMARY

In summarizing I would lay emphasis on the fact that most of the delay in the progress of tuberculosis treatment in the past has been due to a lack of knowledge and indifference, the dissipation of which has emanated largely from discoveries in experimental laboratories and clinics. As a result of the newer knowledge, the early lesions may be seen on the x-ray ten years before clinical symptoms; the tuberculin reaction has become a "plumb line" in dividing all tuberculous infections from non-tuberculous diseases; the finding of bacilli has become a refined science and is the only absolute criterion of diagnosis; and careful pathological studies answer most of the old unknown riddles that have kept doctors in confusion and impeded progress.

The advances in prevention and treatment have followed as the night follows the day, and what we have learned in the last half century with regard to the treatment and handling of tuberculosis has made it one of the most interesting branches of medicine. The interest concerns not only the disease but also its many complications and associated conditions as well as its many social, economic and public health relationships. And finally, the solution of the problem is not yet complete because, like any other phase of science, there is still more to be learned

than we already know. Physicians are, therefore, admonished to claim their heritage!

RESULTS OF SPUTUM EXAMINATION ON 6,304 CONSECUTIVE PATIENTS AT THE CHICAGO MUNICIPAL TUBERCULOSIS SANITARIUM FROM FEBRUARY 1st, 1934 to OCTOBER 1st, 1938

	Number	Per cent.	Per Cent. of Pulmonary Tuberculosis, exclusive of "Ghon Foci"
Tubercle bacilli found on admission by direct smear and concentration	5,364	85.09	87.53
Tubercle bacilli found by refined methods including cultures and stomach washing	502	7.96	8.20
Negative tubercle bacilli, but positive signs of tuberculosis on x-ray and on history	262	4.16	4.27

	Number	Per Cent.			
Non-tuberculous pulmonary Affections	97	1.52	....	.....	.....
Apparently normal by the time of Admission	..	...	52	0.80	.....
Only "healed Ghon foci"	..	...	37	0.59	.....
Extra-pulmonary tuberculosis of					
Lymph nodes	..	...	5	0.08	.....
Spine	..	...	2	0.03	.....
Peritoneum	..	...	1	0.02	.....
Non-tuberculous diseses	79	1.27	....	.....	.....
Bronchiectasis	..	...	25	0.40	.....
Lung cancer	..	...	16	0.26	.....
Lung abscess	..	...	11	0.17	.....
Lues	..	...	7	0.11	.....
Bronchial asthma	..	...	4	0.06	.....
Hodgkin's	..	...	3	0.05	.....
"Cardiacs"	..	...	3	0.05	.....
Non-tuberculous empyema	..	...	3	0.05	.....
Non-tuberculous pleurisy with Effusion	..	...	2	0.03	.....
One each, bronchitis, chronic Sinusitis, Pneumoconiosis, Ca. of Breast, Non-T.B. Spont. Pneumothorax	..	...	5	0.09	.....
Total	176	2.79	6,304	100.00	100.00

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CARDIAC ANEURYSM

An Evaluation of Its Clinical Features

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Aneurysm of the ventricles has been recognized as an anatomical entity since 1757 (Hunter,[9] Galeatti[7]). Its recognition during life was not attempted until comparatively recent years when several observers considered certain murmurs and other physical signs as suggestive or pathognomonic. Most of these criteria proved inadequate, conclusions being drawn from one or two cases and the fact was ignored that the clinical picture is greatly altered by variations in depth and in location of the aneurysm. Excellent summaries of previous attempts to formulate the clinical syndrome of cardiac aneurysm are presented by Sternberg,[21] Ball[2] and by Parkinson, Bedford and Thomson.[18] This report describes our own observations in 21 cases of ventricular aneurysm found at autopsy together with an evaluation, in the light of this experience, of the more important features described in the literature.

LITERATURE SURVEY

It is not sufficiently recognized that there is a close relationship between sclerosis or occlusion of the coronary arteries and ventricular aneurysm; a fact pointed out by Cohnheim and Schulthess-Rechberg[4] in 1881 and re-emphasized by Sternberg[21] in 1914. Elliott[6] commented on the disparity between the scant literature on cardiac aneurysm as compared with the volumi-

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nous reports on coronary disease. Parkinson, Bedford and Thomson[18] estimate that cardiac aneurysm occurs in about 9 per cent. of all cases of myocardial infarction, and Appelbaum and Nicholson[1] reported that cardiac aneurysm occurred in 57 instances of 150 autopsied cases. A wider dissemination of the knowledge of the frequency with which cardiac aneurysm follows coronary thrombosis or acute myocardial infarction should result in the more frequent recognition of cardiac aneurysm during life.

Valuable diagnostic procedures, applicable in the search for cardiac aneurysm are available if suspicion of its existence has been awakened. The most reliable single diagnostic aid is, without doubt, roentgenological exploration. But this procedure, like most others, has its limitations, particularly if the aneurysm is located at the apex of the heart. Kraus,[13] in 1919 briefly described a roentgenogram which disclosed a bulge near the cardiac apex but he did not recognize its significance until autopsy disclosed an aneurysm of the left ventricle in that region. Jacksch-Wartenhorst[10, 11] was the first to recognize cardiac aneurysm roentgenologically and to have the diagnosis corroborated by autopsy later, but priority actually belongs to Sezary and Alibert[19] who described their roentgenological findings three years earlier. Their description and illustrations leave no doubt that their diagnosis was correct even though their findings were not substantiated by an autopsy. Heitz and Corone[8] furnished further valuable roentgenologic information and suggested the oblique positions for those cases in which the aneurysm might be located on the anterior aspect of the heart. The essential roentgenological features have been adequately described by Parkinson, Bedford and Thomson;[18] Steele;[20] Parade;[17] Heim de Balsac[2] and by others and, hence, need not be repeated here. Emphasis should, however, be laid on the fact that fluoroscopic examination with gradual rotation of the patient in the various diameters is indispensable. The greatest difficulties will be encountered when the aneurysm is at the apex, by far its most frequent location. Special measures to visualize this region will be necessary, such as examination during maximum inspiration or after ingestion of some effervescent mixture in order to inflate the stomach. Many aneurysms at the apex will escape recognition even when such precautions are taken; hence cardiac aneurysm should not be excluded merely because there is no positive roentgenologic evidence of its existence. This is exemplified by the four patients in our series in whom an ante mortem diagnosis of cardiac aneurysm was made although they showed no definite roentgenological evidence of that condition.

Cardiac aneurysm may not be disclosed roentgenologically because of small size or because of mere excavation of the inner surface of the ventricle without producing bulging of the external surface of the heart. The latter variety may possibly be recognized by the method recently devised of visualizing the inner aspect of the heart with intravenous radio—opaque substances. Other reasons for non-visualization are splinting of the aneurysmal wall by a firm thrombus or by localized thickening of the pericardium. However, the most frequent cause for failure will reside in the usual location of the aneurysm at the apex, where it is easily masked by adjacent structures, particularly by the shadow of the diaphragm. Strauch[22] found the aneurysm at the apex in 39 of 55 instances at autopsy. Appelbaum and Nicholson[1] found the location of 57 anatomically-proved cases of cardiac aneurysm to be as follows: 34 at the apex, ten on the anterior wall of the left ventricle, six at the interventricular septum, five on the posterior wall of the left ventricle, once in the right ventricle and once at the left border of the heart above the apex; the last being the region most favorable for roentgenologic exploration. Roentgenologic examination is thus of value when positive signs are found, but the absence of such evidence does not exclude cardiac aneurysm in a patient who has previously had an attack of acute myocardial infarction.

Physical signs, when present, furnish valuable diagnostic aid but they are less reliable than roentgenologic evidence. A very significant sign, when found, consists of a circumscribed area of precordial pulsation, palpable just to the left of the sternum and mesial to the region of the apex beat. We did not encounter this sign in our series but examples have been reported by Strauch,[22] Libman and Sacks,[14] Medlar and Middleton,[16] Ball,[2] Parkinson, Bedford and Thomson,[18] Dressler[5] and others. This sign should occur comparatively infrequently since it is best developed when the aneurysm involves the anterior aspect of the heart well above the

apex. However, it has been observed when a large aneurysm of the posterior wall pushed the heart up against the anterior chest wall. In one of our patients an aneurysm at the apex was strongly suspected because of a well-defined, strong apex beat in the presence of moderate cardiac failure seven years after an attack of

Photograph of specimen from case A.G. showing extensive myocardial infarction of left ventricle and localized aneurysm at apex with calcification of the inner surface of the sac.

coronary occlusion. No roentgenological evidence of cardiac aneurysm could be found on repeated periodic examinations during those years but autopsy revealed an aneurysm at the apex as large as a walnut.

Murmurs have long been considered significant but recent observations have cast considerable doubt on their value in the diagnosis of cardiac aneurysm. Murmurs may be of value if they develop after an attack of myocardial infarction. Medlar and Middleton[16] suggest that such murmurs may result from distortion of the mitral ring if the aneurysm is situated at the base of the heart thus producing a "functional" mitral insufficiency, the valves themselves remaining uninvolved. Lutembacher[51] found adhesions between the apex of the heart and the diaphragm which, he states, can also interfere with adequate closure of the mitral valves by the pull exerted during systole. It is conceivable that a similar effect may occur if the aneurysm is located at the base of a papillary muscle, resulting in an out-pouching of the thinned ventricular wall at that point during ventricular systole, dragging with it the papillary muscle and corresponding chordae tendinae. Other factors in the possible causation of murmurs are the passage of the blood stream over the roughened surface of a mural thrombus located in the aneurysmal sac or turbulence set up within the outpouching. It cannot be overemphasized, however, that murmurs may arise coincidentally after myocardial infarction from relative mitral insufficiency or causes other than those inherent in the formation of the ventricular aneurysm. A loud systolic murmur developed at the apex in one of our patients after the formation of an aneurysm in this region; but eight of the 21 patients had no murmurs at all and the remainder showed murmurs either at the apex or base and without apparent regard to the location or size of the aneurysm.

The vast majority of patients with cardiac aneurysm reported in the literature had normal blood pressure. This corresponds with our findings and speaks for the probability that hypertension is not a necessary prerequisite for the development of aneurysm; such development being more closely allied with the extent of the infarction and the general condition of the myocardium. Some patients with cardiac aneurysm were known to have had hypertension before myocardial infarction but the blood pressure fell after the attack and remained at a much lower level, sometimes within the normal range. The significance and mechanism of normal or nearly normal blood pressure in cardiac aneurysm are not yet established but it appears that such a finding is of significance in the diagnosis of this condition.

Electrocardiographic studies have revealed various abnormalities but these are of no definite aid in the recognition of cardiac aneurysm and can be explained on the basis of the antecedent myocardial infarction and the resulting myocardial changes. Electrocardiography may be of value in disclosing a previous coronary

accident and in locating the probable site of the resulting aneurysm on either the anterior or posterior aspect of the heart. Development of heart block may speak for localization in the upper part of the interventricular septum but the electrocardiographic localization will be most accurate if the patient has had only one myocardial infarction.

Another reported finding is the presence of clinical or roentgenological evidence of localized pericardial adhesions, usually near the apex, for which no cause except an antecedent attack of myocardial infarction can be determined. A forcible apical thrust when associated with a faint first heart sound at the apex has been considered a significant combination but was not a striking syndrome in our patients.

Our experience, as exemplified by the following case report, has been that the diagnosis of cardiac aneurysm will rest chiefly on a strong suspicion of its frequent existence after an antecedent attack of myocardial infarction, especially when the blood pressure is normal or nearly normal and on the presence of some of the other signs previously described. This presumptive diagnosis can be corroborated in many instances by adequate roentgenological exploration, but failure to find such positive roentgenological evidence does not exclude the possibility that cardiac aneurysm is present.

CASE REPORT

A. G., a male 69 years of age had been in excellent health up to about eight years before admission to the Michael Reese Hospital. His blood pressure had been normal, and periodic examinations disclosed no abnormal findings until about seven years ago when he developed a severe attack of coronary thrombosis. He remained at bed rest for three months after which he was in comparative comfort for about five years, during which time he was moderately active. Precordial pain and evidence of severe congestive failure recurred about three years ago at which time he developed edema, encapsulated pleural effusion just lateral to the angle of the left scapula and enlargement of the heart downwards and to the left. The pulse was regular and of good quality; the systolic blood pressure was 140/90 mm. of Hg. The heart sounds were of good quality but a loud systolic murmur was now present at the apex, transmitted best toward the base of the heart. Roentgenological examination in three diameters confirmed the findings on palpation and percussion but revealed no localized distortion of the cardiac shadow. The electrocardiogram showed a rate of 91, regular sinus rhythm, QRS of low amplitude and slurred in the limb leads, P-R interval 0.16 seconds and T small in lead I. The S-T segments were slightly depressed in leads II and III and the precordial lead showed nothing abnormal. He improved after six months of bed care, after which he was again in comparative comfort for nearly three years during which time he was under constant ambulatory management. He was readmitted to the hospital on February 20, 1939 with an obvious bronchopneumonia. Examination at this time revealed in addition, marked icterus, a blood pressure of 130/90, rapidly progressive congestive heart failure and, within a short time, signs of mental confusion and coma. An electrocardiogram taken on February 23rd showed a rate of 79, P-R 0.16 second, QRS of low amplitude and slurred in the limb leads, iso-electric T in lead I and a few auricular extrasystoles. Nothing abnormal was seen in the precordial leads. Moderate digitalization for a week was followed by another electrocardiogram which showed the changes mentioned before, and in addition, slight depression of the S-T segment in leads I and II and a nodal parasystole with intereference dissociation. There was no change in the physical findings and a portable anteroposterior roentgenogram showed changes consistent with bronchopneumonia and pulmonary congestion but no evidence of either pulmonary infarction or cardiac aneurysm although special efforts were taken in an attempt to disclose the latter. The patient died on March 10th and autopsy performed by Dr. O. Saphir revealed generalized arteriosclerosis, advanced coronary arteriosclerosis, marked narrowing of the anterior descending branch of the left coronary artery and an organized thrombus in the circumflex branch of the left coronary artery near its point of origin. The heart weighed 475 gm., the epicardium was normal everywhere but the myocardium of the left ventricle near the apex was very thin and bulged outward, forming a sacular cavity about 3 cm. in diameter. There was also thinning and bulging of the lower anterior portion of the ventricular septum and, to a lesser degree, of the posterior aspect of the left ventricle at the apex. The endocardial surface of the aneurysmal sac showed a large area of calcification but contained no thrombus. Areas of myocardial fibrosis were present in other regions and histological examination revealed marked fibrosis and degenerative changes of the muscle fibers. There was no evidence of pulmonary infarction and the biliary tract was normal.

SURVEY OF PRESENT SERIES

Cardiac aneurysm was recognized clinically in four instances in our series of 21 cases seen at autopsy chiefly because the condition was strongly suspected rather than on the basis of positive clinical or roentgenological evidence. More would have been diagnosed, we feel certain, if the possibility of its occurrence had been considered in the other patients.

Twenty of the twenty-one patients were males. Our series is too small to be of statistical sig-

nificance but these findings correspond in general to other reports in the literature. The age grouping corresponds to that expected for the underlying causative factor. Five of our patients were between the ages of 40 and 50, five between 50 and 60, six from 60 to 70, four from 70 to 80 and one, a suicide, was 90 years of age. The fact that so many of these patients attained such ages suggests that cardiac aneurysm is not a serious menace to life. Sufficient emphasis has not been laid in the past upon the fact that multiple cardiac aneurysms is not an uncommon finding in the same patient. Six of our 21 patients had two or more such aneurysms. This is not surprising since the underlying coronary disease is progressive and favors the formation of subsequent myocardial infarctions. Multiple cardiac aneurysms, like single ones, do not appear to affect the prognosis.

Syphilitic changes in the aorta and aortic valves were found in only one patient, an observation which accords with the views expressed by others on the minor role played by this infection in the production of cardiac aneurysm. Arteriosclerosis of the coronary arteries was well developed in all of the 21 patients including the one in whom syphilitic changes were found. Complete occlusion of a coronary artery was absent in four instances, a finding which supports the argument that complete closure is not necessary for the occurrence of myocardial infarction and subsequent aneurysm and that the clinical diagnosis should read recent or recurrent "myocardial infarction" rather than coronary thrombosis. No adequate explanation has as yet been given for the development of myocardial infarction in the absence of complete coronary obstruction and the question of coronary spasm or other form of physiological coronary insufficiency may be raised. A single occlusion was found in ten instances. Other branches of the coronary arteries in these ten instances, however, showed areas of constriction in several places. This suggests the speculation as to whether such a single occlusion combined with restriction of the lumina in other branches of the coronary arteries per se is sufficient to explain myocardial infarction and resultant aneurysm or whether such lesions may be the result of additional reflex spasm of coronary branches comparable to that which occurs in embolism of the pulmonary or peripheral arteries. Evidence of such a mechanism would, of course, open a new therapeutic approach in myocardial infarction. However, we are aware that these findings in the arteries may prove inaccurate since detailed examination after injection of the vessels was not made in these specimens. Complete thrombotic closure of the right coronary artery was present in three patients without the occurrence of aneurysm in the right ventricle although the left coronary artery in these instances also showed some sclerosis and narrowing. This is in sharp contrast to the results when the left coronary artery is occluded. This difference may be due in great part to the fact that adequate blood supply may be possible via the thebesian channels which are so profuse in the right ventricle and comparatively infrequent in the left.[12] Involvement of the left coronary artery thus appears to be a much more serious affair than disease of the right. This observation is also in accordance with the reports in the literature, to the effect that aneurysm of the right ventricle is much less frequent than of the left. It has also been observed that when an aneurysm did develop in the right ventricle after occlusion of the right coronary artery the sac was usually at the base rather than near the apex.

The reports in the literature that blood pressure is almost always within the normal range when cardiac aneurysm has developed is in agreement with the findings in our patients. Hypertension was present before or after myocardial infarction in ten of our 21 patients, but definite hypertrophy, considering a heart weight of 375 gm. or more as a criterion, occurred in 20 instances. The absence of hypertension in 11 of our 21 patients supports the contention that heart failure alone may result in cardiac hypertrophy and that it is not necessary to assume that hypertension must have existed in order to explain the hypertrophy. One may also question whether hypertrophy affords a measure of protection against the formation of cardiac aneurysm, a doubt which is heightened by the fact that hypertrophied myocardium is no longer normal muscle tissue.

Our experience does not agree with reports in the literature that a forcible apex beat and faint first heart sound is a combination of great value in the diagnosis of cardiac aneurysm. It is possible that such an association does occur but faint heart sounds were found in only 12 of

our 21 patients and the change could be explained in some of these on the basis of generalized myocardial disease rather than on cardiac aneurysm as such. As already mentioned, murmurs were of no diagnostic significance in our patients and in eight of our patients no murmurs were audible.

Of great significance was a history or evidence of an antecedent myocardial infarction. Seventeen of our patients gave a definite history but two were positive that they never experienced pain or other suggestive symptoms. No history was obtained in one, a suicide, and the history in the remaining patient was unreliable. Only eight patients were aware of more than one attack, which belies the assumption that a history of multiple attacks of myocardial infarction is necessary for a diagnosis of cardiac aneurysm.

The duration of bed rest after an attack of myocardial infarction was not specified by 11 of our patients but seven remained in bed for periods ranging from two days to two weeks; two additional patients were not put to bed rest at all because they experienced no pain and only one of our patients was known to have remained in bed for more than six weeks. These observations suggest that inadequate bed rest may be an important prerequisite of cardiac aneurysm. This is in accord with the experience of Sutton and Davis[23] that cardiac aneurysm was more likely to develop in animals which were exercised shortly after myocardial infarction was produced. More clinical observations are necessary before a definite conclusion of this relationship can be drawn. Certainly, the magnitude of the infarct and the condition of the myocardium are also important factors. Nevertheless, our observations are very suggestive.

The lack of assistance obtained from roentgenological examination in our series is, to a certain extent, due to inadequate use of it. We believe that more of the aneurysms would have been recognized had we subjected our patients to more adequate roentgenological exploration. The roentgenological examination should be made in all diameters with special attention to the region of the apex in all cases who formerly had an attack of myocardial infarction. The average diameter of the aneurysms observed by us varied from 2 to 4 cm. and the average depth from 1.2 to 3 cm. Methods devised to visualize the inner aspects of the heart should certainly aid in disclosing abnormalities of such magnitude and outward bulging of such extent should also be recognized unless obscured by adjacent tissues or by unfavorable roentgenological localization. It is reasonable to suppose that the deformity is larger during life when the elevated systolic, intracardiac pressure may aid in detection of this less resistant area of the ventricular wall. It should be noted, however, that the foregoing measurements are of the inner aspects of the aneurysmal sac and are probably smaller than the outward bulging. But even the external bulging must compare favorably with the size of a niche resulting from peptic ulcer of the stomach or duodenum. Roentgenological recognition by ordinary methods thus becomes difficult only if the outward bulge is small or absent or if it is located at the apex. The most favorable location for diagnosis is at the left border of the heart above the region of the apex or on the anterior aspect when the right or left oblique diameters are used. It may be mentioned in passing that the aneurysm was located at the left border of the heart above the apex in only one of our patients. The foregoing difficulties emphasize the fact that roentgenological exploration yields valuable information only when positive evidence is found but it is equally true that absence of such manifestations must not exclude a diagnosis of cardiac eneurysm.

Rupture of the aneurysm is very rare; it did not occur in our series and cardiac failure cannot be attributed as a direct result of the aneurysm. Six of our patients lived in comparative comfort for from one and one-half to two and one-half years and one was fairly active for about seven years. Personal communications from those who have had some experience with cardiac aneurysm agree with our concept that this condition does not alter the prognosis. The prognosis of cardiac aneurysm is in all likelihood decided by the general condition of the myocardium and by the future developments in the coronary arteries. Cardiac aneurysm should be recognized not for its prognostic significance but in order that its manifestations may be properly evaluated. Cardiac aneurysm requires no special therapeutic measures other than those directed to the underlying cause and to the general condition of the heart. But this study does emphasize the necessity of adequate bed rest after myocardial infarction in order to minimize the

development of the acute stage of cardiac aneurysm.

CONCLUSIONS

1. A study was made of 21 patients in whom cardiac aneurysm was found at autopsy and in whom the clinical course was followed closely until the time of death. A correct diagosis was made in only four instances but this was due in great part to the fact that the frequency with which such an aneurysm follows myocardial infarction was not appreciated.

2. Twenty of our 21 patients were males and the age group distribution was that seen in myocardial infarction, the usual cause of ventricular aneurysm.

3. Multiple cardiac aneurysms were found in six instances, a fact which should not be surprising in view of the progressive nature of the underlying disease.

4. Arteriosclerosis with complete occlusion of one or both coronary arteries was present in 17 instances and sclerosis without occlusion was found in the remaining four. Syphilitic changes were present in only one patient but appeared to play no part in the formation of the cardiac aneurysm.

5. Involvement of the right coronary artery, even complete thrombotic closure, did not result in aneurysm of the right ventricle. This was observed in three instances even when concommitant involvement of the left coronary artery was present. Disease of the left coronary artery was always found in aneurysm of the left ventricle.

6. Blood pressure was usually normal and tended to remain at a lower level if marked hypertension existed prior to the discovery of the aneurysm.

7. Cardiac hypertrophy was found in 20 of the 21 hearts although hypertension was not present in ten instances either before or after the attack. This brings up the possibility that factors other than hypertension or similar well known causes need not be present in order that hypertrophy develop.

8. The apparent value of roentgenological evidence and other clinical manifestations are discussed and the importance of adequate bed rest after myocardial infarction is emphasized.

9. Alertness to the frequency with which cardiac aneurysm develops after myocardial infarction will aid materially in diagnosis. Positive roentgenological evidence, when present, is devisive but absence of such findings in no way excludes the presence of an aneurysm.

10. The prognosis of cardiac aneurysm of the type under discussion depends chiefly on the degree of associated coronary and myocardial involvement. No special therapy is indicated other than adequate care of the underlying condition and of the general state of the myocardium.

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CARDIAC REVIEW OF 1939

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1. SYMPTOMS AND SIGNS

The clinical correlation is very important in the interpretation of cardiac sound records, for as Sacks and Roberts[1] found, it is not correct to interpret a few irregular oscillations in systole or diastole as a murmur because they may be due to the impact of the apex against the chest wall or to other extracardiac factors. Shapiro[2] followed 102 patients with nonpathologic murmurs (systolic) over a period of years and the original diagnosis was found to be correct in 98%; the murmurs of nonpathologic nature are most likely to be short and of slight intensity and are not well transmitted.

METHODS OF CARDIOVASCULAR EXAMINATION

1. *Electrocardiography:* Chamberlain and Hay[3] stated that the age variations in the normal electrocardiogram were slight, the most important being an increase in the left axis deviation in the latter decades of life. Strouse, Katz and Binswanger[4] emphasized that while the electrocardiograph is a useful tool in clinical practice, it constitutes only one part of the clinical examination and it must be correlated with the rest of the examination. Wood and Selzer[5] reported a new sign of left ventricular failure in cases of hypertensive heart disease and of aortic incompetency, a widened P wave of low voltage, usually bifid or flat-topped. Geiger[6] stated that although the electrocardiograph is of great value in substantiating and indicating the approximate location of coronary thrombosis, especially if supplemented by chest leads, absolute reliance upon this laboratory aid is inadvisable and should not be substituted for a carefully determined clinical diagnosis. Wood and Selzer[7] feel that multiple chest leads are of value not only because they may yield diagnostic evidence of myocardial infarction, but also because they are a safeguard against faulty interpretation of Lead 4. Ashman[8] concluded that valuable as the precordial lead is, definite electrocardiographic abnormalities appear in the standard leads, with Lead 4 normal, more often than the reverse; therefore, the chest lead, at least as at present employed, cannot be regarded as a substitute for the limb leads. Comeau and White,[9] in a study of 200 electrocardiograms showing right axis deviation, found most commonly mitral stenosis (44.5%) no heart disease (37.5%), congenital (7%), and associated with other conditions (11%; coronary heart disease was found in only seven (3.5%), and in only one was it more than of a slight degree.

2. *Roentgenography:* Kuttner and Reyersbach[10] stated that the chief value of any radiologic procedure in cases of early rheumatic heart disease is to detect changes in the size or contour of the heart in the same patient at different times.

3. *Circulation Time:* In 15 patients who were receiving full doses of digitalis, Wall[11] used calcium gluconate to measure the circulation time 27 times without any clinical evidence of the slightest untoward effect. Bernstein and Simkins[12] found the magnesium sulfate test, 6 cc. of a 10% solution rapidly injected intravenously, well adapted for bedside and office use, but the presence of a high normal circulation time is common in advanced degrees of cardiac decompensation.

3. ETIOLOGY OF HEART DISEASE

1. *Congenital:* Ash and Harshaw[13] reviewed 230 cases of congenital heart disease seen in the Philadelphia Children's Hospital from 1922 to 1936 and noted that the death rate of cyanotic infants was twice that of the noncyanotic and continued to rise progressively with age, whereas that on noncyanotic individuals tended to show only a slow increase after the second year of life. Greenspon and Leaman[14] reported a case of complete pulmonary atresia with hypertrophy of the bronchial arteries simulating the tetralogy of Fallot; death occurred at the age of five years and was attended by congestive heart failure, auricular fibrillation, and possible cerebral thrombosis. Herndon, Vass and Donovan[15] cited a case of the tetralogy of Fallot in a man past 49 years of age who was active and self-supporting until his terminal illness; death was due to sepsis following furunculosis of the nose and cavernous sinus thrombosis; in the terminal sepsis there were crossed or paradoxical emboli which passed through the septal defect.

2. *Rheumatic:* Massie and Levine[16] made a study of 135 patients with acute rheumatic pericarditis whose average age was 19.6 years; 22

(16.3%) died of the acute infection, and in 82 of the remaining 113 cases follow-up data were available after an average interval of seven years. Twenty-five patients died after recovery from the initial infection at an average interval of 6.7 years. It was very striking that 36% of those examined on follow-up had no evidence of organic heart disease and an additional 14% had only mitral insufficiency. Even after a desperately severe attack of acute pericarditis recovery was often complete. Their study emphasized the great importance of injury to the valves of the heart in the subsequent prognosis of acute rheumatic infections and the minor role that the heart muscle or pericardium plays after recovery occurs from acute infection. Bland and Jones,[17] from a ten-year follow-up study of 314 children and adolescents with potential rheumatic heart disease, showed that (1) characteristic signs of permanent valvular deformity not present at the time of the original illness have appeared later in 79 patients (25%); (2) that this so-called delayed appearance of heart disease was clearly associated with coexisting signs of recurrent rheumatic fever in two-thirds of the group; and (3) in the remaining one-third it appeared insidiously.

3. *Bacterial:* The study of 87 cases of bacterial endocarditis by Middleton and Burke[18] offers material support to the importance of the diagnostic triad of petechiae, splenomegaly, and a positive blood culture for Streptococcus viridans. Buchbinder and Saphir[19] noted heart failure of a fairly advanced degree in 18 of 40 patients with subacute bacterial endocarditis, a clinical frequency of 45%, and the combined frequency of heart failure for the groups studied pathologically and that studied clinically was 75%. Capps[20] reported a follow-up of 139 cases of subacute bacterial endocarditis, in all of which there were heart murmurs, fever, blood cultures yielding Streptococcus viridans, and often embolic phenomena; and there were 11 cases who survived more than five years after the onset of infection. A comparison of the recovered with the fatal cases showed that in the recovered group the fever on the whole was lower and embolic phenomena less in evidence; none developed cerebral emboli or Osler's nodes; clinically the recovered patients with few exceptions were up and around complaining of fatigue, palpitation, sweating, loss of appetite and loss of weight. Spink and Crago[21] believe that sulfanilamide and its related compounds will be of doubtful value in the treatment of patients with subacute bacterial endocarditis because of the very nature of the focus of infection. The proliferating mass of bacteria situated well beneath the surface of the vegetations is probably protected, at least in part, from the action of the free sulfanilamide in the blood, as well as in specific antibodies. Allen[22] concluded, contrary to the long established current concept, that the bulk of a vegetation of acute or subacute bacterial endocarditis is derived from components of inflamed and fibroplastically deformed valves and is not derived from the blood flowing over the valves. De Navasquez[23] stated that in Streptococcus viridans endocarditis, pyemic abscesses in the myocardium or elsewhere are usually absent and embolic lesions, when encountered, do not usually suppurate. Allen[24] found that in from 50% to 75% of cases bacterial endocarditis is superimposed on rheumatic endocarditis, which commonly produces a valvular fibroplastic deformity with stenosis; this lesion takes the form of a projecting shelf or barrier, usually at the line of closure, against which the blood stream strikes. By virtue of this obstruction to the systolic discharge, the site of the deformity suffers a distinctly greater impact and contact than the normal valve leaflet; this contact with the blood (and organisms in a bacteremia) is further enhanced by the diastolic backflow due to insufficiency—the usual concomitant of stenosis. Polowe[25] stated that splenectomy has been performed in four cases of proved bacterial endocarditis, two of which were his own; one patient is alive and well more than 20 months after splenectomy. Orgain and Poston[26] reported a case of gonococcal endocarditis with recovery after sulfapyridine. Forster[27] cited two cases of fatal bacterial endocarditis due to Salmonella suipestifer, Uhr[28] reported a case of bacterial endocarditis, clinically and pathologically undistinguishable from an ordinary case, due to the Actinomyces bovis. Smith and Curtis[29] analyzed another case of brucellosis with aortic vegetative endocarditis, refractory to three courses of sulfanilamide. Rakov[30] reported a case of subacute bacterial endocarditis caused by the type XVIII pneumococcus.

4. *Syphilitic:* Venzoni[31] found that among 199 cases of syphilitic aortitis sudden death had

taken place in 39, in which it was noted that stenosis of the coronary arteries (16 cases), aortic insufficiency (four cases), or coronary stenosis in association with valvular insufficiency (11 cases) existed. Welty[32] encountered syphilitic cardiovascular disease in 6.93% of 15,00 autopsies at the Philadelphia General Hospital; the incidence of syphilitic aortic regurgitation was 1.44% and of aneurysm 1.28%. Thompson, Comeau and White[33] analyzed the data on 260 indivduals who contracted syphilis 15 to 25 years before the study was made with respect to the present cardiovascular status of the subjects and the relationship of treatment thereto; their study gave some support to the clinical impression that adequate treatment of syphilis tends to prevent the late clinical manifestations of cardiovascular syphilis.

5. *Myxedema:* Savy, Froment and Jeune[34] showed that two types of cardiovascular disturbances are observed in myxedema; to the first group belong those which can be traced directly to the myxedematous condition and which subside or disappear under the influence of organotherapy with thyroid. In the order of their frequency they are the diminution of voltage of the electrocardiographic tracings, more or less severe degrees of cardiac hypertrophy, some disturbances in tension, and, in exceptional cases, anginal symptoms. All of these disturbances are benign and easily cured if they are not of too long standing. To the second type belong the lasting and incurable forms of cardiovascular disturbances which are often aggravated rather than improved by thyroid therapy. Their prognosis is unfavorable and the connection with the thyroid insufficiency is uncertain or indirect. Marzullo and Franco[35] reported a case of severe myxedema in which there were the usual signs of myxedema heart and polyserous effusions, with the 48-year-old female patient under treatment for ten years and who continues to be well.

6. *Hypertension:* Vayl[36] selected for microscopic study 61 cases in which death resulted from hypertensive disease not complicated by arteriosclerosis or atherosclerosis of the coronary arteries. This series contained a group of 14 hypertensive patients from 20 to 38 years of age and 47 patients from 40 to 68 years of age. A microscopic study with various staining methods was made of portions of the heart, upper sympathetic plexus, the cervical ganglion of the vagus, the stellar ganglion and the solar, thoracic, and lumbar plexuses, from which he concluded that the primary phenomenon in hypertensive disease is the rise of the blood pressure and vascular spasm. La Place[37] stated that hypertensive heart disease is a common cause of sudden death which, however, seldom occurs without conspicuous warning. Routier and Gerbeaux[38] summarized their obesrvations and reported that in 38% of hypertensive patients examined, the electrocardiogram was either normal or showed a simple deviation to the left, but in 62% there were strong pathologic changes; and of the latter group 75% showed an inversion of the T wave in the first lead.

7. *Pulmonary:* Chronic pulmonary disease, according to Griggs, Coggins and Evans,[39] is an important cause of cor pulmonale, but the various pulmonary lesions vary greatly in their effect upon the heart; pneumoconiosis showed the highest percentage, emphysema ranked second, and pulmonary tuberculosis ranked lowest in the incidence of cor pulmonale. It seems clear, stated Chapman, Dill and Graybiel,[40] that persons with severe thoracic deformities, particularly those with a right-sided dorsal kyphoscoliosis, suffer an habitual dyspnea that may increase to the point of imposing a severe limitation of activity; and later on in life palpitation, fainting attacks, or aggravation of the dyspnea by exertion or changes in position may harass those deformed. Such a sequence of events is sufficient to diagnose pulmonocardiac failure, and the average duration of life is only thirty years. Giering and Charr[41] studied 25 miners, of which 16 had anthracosilicosis without pulmonary tuberculosis, and nine had either moderately advanced or far advanced tuberculosis in addition to the anthracosilicosis; yet they ranged in age from 36 to 67 years and the duration of their occupation as miners, before cardiam symptoms appeared, ranged from three to 40 years.

8. *Coronary:* Clawson[42] analyzed 928 cases of coronary sclerosis from which he stated that coronary sclerosis of the atherosclerotic type, with or without thrombosis and infarction, is the most common cause of sudden cardiac death; and that most patients die while at rest and only a relatively small number during effort. Paterson[43] holds that the formation of coronary thrombi is a gradual process, sometimes occupying several days before occlusion of the coronary lumen with

its resulting cardiac pain is produced; therefore, he concluded, the activities of a patient immediately preceding the onset of an attack of coronary thrombosis have no relation to the etiology of the precipitation of a thrombus but are purely coincidental. Gordon, Bland and White[44] found the relative incidence and the degree of coronary atherosclerosis to be significantly greater in the 600 patients from the private departments than in the 2,800 patients from the general wards of the Massachusetts General Hospital; the greatest difference occurred in the middle-aged patients, in whom coronary occlusion was found to be twice as frequent in the private group. Starr[45] stated that there seems to be no causal relationship between coronary thrombosis and cholelithiasis, although disturbances of cardiac rhythm may well be due to gallbladder dysfunction. Weiss[46] noted an early rise of blood pressure during the early painful stages of coronary occlusion, and added that this should not be confused with the increase in pressure which often accompanies an attack of angina pectoris. Goodson and Willius[47] studied 30 cases of coronary thrombosis among persons less than 40 years of age and felt that the report is justified in again emphasizing that this disease is occurring with increasing frequency among young persons. Scott and Garvin[48] reported a characteristic case of rupture of the infarcted interventricular septum as the signs were those of a mycardial infarction plus those of an interventricular septal defect. Brumm and Willus[49] studied 257 patients with severe coronary disease who underwent necessary surgical operations, of which 11 (4.3%) died from cardiac causes; death resulted from coronary thrombosis in seven, from congestive heart failure in two, and abruptly without apparent thrombotic occlusion in two. Weiss[50] described three cases of acute coronary occlusion complicated by hiccups; the cause was obscure, the treatment disappointing, and they had no prognostic significance.

9. MISCELLANEOUS

(A) *Trauma:* Boas[51] stated that when a person previously free from symptoms of coronary artery disease has such symptoms after an injury to the chest or unusual physical exertion, the heart disease may be attributed to these external factors; when a person with angina pectoris of the simple type has coronary artery occlusion following such injury or effort, the sudden progression of the lesion may be ascribed to these causes; and when such a sequence of events occurs in a person gainfully employed while he is at his task, it is proper to conclude that the cardiac disability is compensable, provided the cardiac symptoms accompany or follow immediately on the event of which the cardiac injury is ascribed. A case of contusion of the heart, with recovery, in a 17-year-old boy, was reported by Smith and McKeown,[52] and although the electrocardiograms presented a pattern that might be associated with pericarditis, in this case the primary damage was to the myocardium and the nonbacterial pericarditis was secondary; the contusion of the myocardium was produced without evidence of any damage to the chest wall. O'Farrell[53] reported the case of a 59-year-old male who, while driving an auto, was involved in a head-on collision with another car and developed severe precordial pain and became short of breath; later auricular fibrillation developed and death occurred suddenly three months after the accident due to cardiac failure which was caused by degeneration of the heart muscle with consequent dilatation (aneurysm) of a portion of the left ventricle. Through a questionnaire sent to the members of the American Association of Thoracic Surgery, the American Surgical Association, and the Southern Association, Bigger[54] found that of 141 patients with heart wounds operated upon by members of these associations, 71 recovered and 70 died. Crynes and Hunter[55] stated that commonly, pericardial rupture is merely one of many injuries and in most instances contributes little or nothing to the cause of death.

(B) *Pregnancy:* Active rheumatic fever, according to Cohen and Thomson,[56] is not a significant factor contributing to heart failure during pregnancy and is notably rare at that time. Page and Campbell[57] reported three cases of Streptococcus viridans endocarditis in pregnancy and emphasized that the disease may be easily mistaken for pyelonephritis or puerperal sepsis at the onset but is readily differentiated by culture of the blood, examination of the heart and careful search for embolic phenomena. Interference with the pregnancy or operative delivery is not advisable, and efforts should be made to carry the fetus to viability. Reid and Teel[58] believe that the distressing pulmonary edema

often encountered in severe and fatal eclampsia is the result of left ventricular failure, and for this reason rapid full digitalization of all eclampsia patients is indicated. Scholder[59] reported the case of a 33-year-old woman who showed mitral insufficiency and stenosis with auricular fibrillation that developed after the first pregnancy; frequent electrocardiograms from then on (1932) showed a complete heart block with a rate of 48. She was delivered at eight months by Cesarean section because of premature rupture of the bag of waters, and two years later the electrocardiogram still shows complete heart block.

(C) *Others:* Hegglin[60] found that the combination of diabetes mellitus and heart disease is comparatively frequent, as the heart does not escape the general damage that is produced in the body by the severe metabolic disturbance; this applies chiefly to aged persons with diabetes. From a postmortem study of coronary atherosclerosis in diabetes mellitus, Root et al.[61] found coronary occlusion much more frequently in diabetic than in nondiabetic persons, the difference, for example, in the age group from 40 to 60 being 23% for the diabetic as compared with 6% for the non-diabetic patients. Ellis and Faulkner[62] emphasized that patients with anemia are often erroneously diagnosed as suffering with heart disease, and also that since anemia is an aggravating burden in persons with organic heart disease it is important that it be recognized and treated in such cases. Perry[63] described the cases of three children who recovered from attacks of diphtheria with persistent conduction defects; in two this took the form of complete heart block and in the third of bundle branch block, the lesions developing at the time of the diphtheria and persisting for many years. Whitehill, Longcope and Williams,[64] from a study of 138 patients, mostly young adults, suffering from the form of acute hemorrhagic nephritis that follows directly from an active infection due to the hemolytic streptococci usually, observed that circulatory insufficiency often forms an integral part of the illness. It is characteristic that evidences of circulatory failure may appear at the onset or during the first few days of the disease; it may well lead to an increase of the subcutaneous edema, or to cerebral or pulmonary edema, and may produce other deleterious effects such as interference with the function of the injured kidneys. These conditions are uncommon in mild attacks of nephritis, frequent in moderately severe attacks, and present in the majority of cases of severe acute nephritis; the use of digitalis in full doses has seemed to benefit some of these patients with severe circulatory failure. Jones and Bramwell[65] reported the case of a bar attendant with a history of chronic alcoholism and gross dietary deficiency, with anorexia, vomiting, epigastric pain, extensive edema, a greatly enlarged heart, gallop rhythm and tachycardia, who was treated with parenteral vitamin B_1. He lost his edema and other signs of cardiac failure in five days, the heart diminished greatly in size, and he was discharged cured in 17 days after the admission.

4. PATHOLOGY

Myocardial: Berk[66] showed the significant clinical features of cardiac aneurysm to be a history of coronary thrombosis with congestive failure, cardiac enlargement, a weak first heart sound, and a diffuse heaving precordial impulse. Fawcett and Ward[67] reported a cardiac myxoma in a patient which obstructed the mitral orifice and produced murmurs like those of mitral stenosis, fainting attacks related to posture, and sudden and unexpected death. Scott and Garvin[68] noted that the development of congestive failure without other apparent cause, in a patient with malignant disease, was the most important clinical finding pointing to cardiac metastasis. Martin, Tuohy and Will[69] reported another instance of a primary malignant tumor producing extreme diffuse stenosis of the pulmonary artery at its orifice; it was a polymorphous cell sarcoma which invaded the wall of the pulmonary artery to, and beyond, its right and left branches.

Endocardial and Valvular: Texon[70] stated that calcific aortic stenosis is not uncommonly followed by sudden death, that Adams-Stokes attacks may occur, and that its presence in an aortic lesion when no mitral lesion is present should be borne in mind. According to Dry and Willius[71] cardiac hypertrophy is present in a high percentage of cases of calcareous disease of the aortic valve and closely parallel the degree of stenosis. Friedberg and Sohval[72] made a study of 15 cases of calcific aortic stenosis in which detailed pathological examination had shown that the lesion was non-rheumatic, in four of which there was an associated syphilitic aortitis. These cases fell into three groups; the first,

constituting five cases, was characterized by the development of left- and right-sided heart failure, whose history revealed the typical symptoms of calcific aortic stenosis, including angina pectoris, dizziness, and syncope, and in occasional cases death occurred suddenly; in the second group, also consisting of five cases, the valvular lesion was discovered accidentally in patients who died of an unrelated disease; in the third group of four patients, which were segregated because there was an associated syphilitic aortitis and aortic valvular disease, there was a close resemblance to the first group both in the occurrence of cardiac failure and in the appearance of other characteristic symptoms.

Pericardial: Stewart and Heuer[73] suggested that the presence of chronic constrictive pericarditis should be considered when there are signs of congestive heart failure in the absence of the usual causes of failure; their experience leads them to recommend pericardial resection in these cases. For in the nine cases which they[74] have studied, seven have been subjected to the operation of pericardiectomy and all of the patients have recovered from operation; three patients are cured in the sense that their symptoms and signs have disappeared and they are able to lead normal active lives; three patients are markedly improved and one is improved, although sufficient time has not elapsed to evaluate the results of operation. Warburg[75] described five cases of "pure" constrictive pericarditis treated operatively, with good results in three cases and no deaths. Hunter and East[76] gave the results of pericardiolysis in three patients, of whom two had developed chronic constrictive pericarditis (Pick's syndrome) about a year after acute pericarditis and the third showed on radiography calcification in the pericardial sac, although the syndrome had not developed. All three patients had pleurisy with effusion after operation; and of the patients with Pick's syndrome, one is apparently cured and the other only partly relieved. Burchell et al,[77] presented evidence that the electrocardiographic picture of acute pericarditis is attributable to subepicardial myocarditis. Ross[78] emphasized that it is dangerous to try to treat cases of suppurative pericarditis by repeated aspiration. Orgain and Poston[79] reported a case of meningococcic infection producing pericardial effusion with cardiac tamponade in a 32-year-old white male; complete recovery followed pericardial paracentesis and treatment with sulfanilamide and antimeningococcic serum. Rajasingham[80] described the case of a 20-year-old female who received a blow on the front of the chest and was acutely ill the next day; no external injuries were noted, but she had a massive traumatic hemopericarium and treatment by aspiration was successfully carried out.

Vascular: Rogers'[81] reference to published accounts of 30 authentic cases of dissecting aneurysm of the aorta shows a rapid increase in the frequency with which the condition is being recognized; one of these patients lived 27 months after the acute attack. Rottino[82] studied medial degeneration in the aorta in 12 selected cases of dissecting aneurysm of the aorta; the lesion was characterized by loss of muscle, elastic tissue and collagen, lack of inflammatory reaction and healing by loose scar formation and by regeneration of muscle and elastic tisue; the loss of muscle appeared to be the initial lesion, and the degeneration of the remaining elements follows. Roberts[83] reported the 28th case in which there were typical changes of medionecrosis aortae idiopathica cystica, as described by Erdheim; in all of them, this disease was the pathogenic basis for the ruptured dissecting aneurysm of the aorta. Horn, Dack and Friedberg[84] studied a group of 42 cases of embolism of the pulmonary artery, in eight of which recent structural changes in the myocardium ordinarily resulting from acute myocardial ischemia were revealed; the factors necessary for the production of such myocardial changes are shock, asphyxia, and exaggerated vagal reflexes resulting from obstruction of the pulmonary arteries. These factors, alone or in association, lead to insufficiency of the coronary circulation. Maurer[85] reported two cases of aneurysm of the aortic arch with absence of the pulse in the versels of the upper extremities and neck so that the clinical picture was that of cerebral disease, with attacks of dizziness and syncope. Gross and Hubbard[86] reported the first successful case of surgical ligation of a patent ductus arteriosus in the hope of preventing subsequent bacterial endocarditis and with the immediate purpose of reducing the work of the heart caused by the shunt between the aorta and pulmonary artery. Later Gross[87] stated that the operation had been successfully performed on

four patients. The second successful case[88] was also reported in detail.

5. FUNCTIONAL DISORDERS

Congestive Failure, Including the Use of Digitalis and Diuretics: Gavey and Parkinson[89] stated that digitalis is always indicated in congestive heart failure, irrespective of rhythm, but it is often inefficient, as it fails completely in about one-third of all cases; in heart failure with normal rhythm, digitalis is helpful in more than 50% of cases. Blumgart and Altschule[90] reported that digitalis in doses sufficient to induce therapeutic effects may be given to patients with partial heart block without causing interference with the orderly passage of impulses from the auricles to the ventricles. Bedford[91] stated that in certain kinds of heart disease the left ventricle fails before the right, causing pulmonary and pleural congestion without dropsy or systemic engorgement. Plotz[92] noted that wheezing respiration is common in left ventricular failure; it may occur without basal rales, in which case it closely simulates allergic asthma in symptoms, physical signs and response to adrenalin. Smith et al.[93] found that a comparison of the fatal dose and of the concentration of calcium in the serum at death in normal and digitalized dogs indicated that, by the type of experiment which they described, the lethal effects of calcium and digitalis are neither synergistic nor even completely additive. The toxic effects of urginin (squill), according to Vander Veer et al.,[94] were like those of digitalis and there was no clinical evidence that the drug was less toxic than digitalis when used in sufficient dosage; in patients with severe cardiac damage who developed toxic rhythms after digitalis therapy, similar disturbances in rhythm followed the use of urginin; and in patients who have been digitalized or who have received digitalis recently, urginin must be used with the same care with which the additional digitalis would be used, as its effect is cumulative with the digitalis already given. Brams and his co-workers[95] do not advocate strophanthin instead of digitalis in the routine management of cardiac failure; however, it is their impression that it is a safe and rapidly acting drug when used in proper dosage and in suitable patients. The properties of strophanthin are practically those of digitalis, but its speed of action and safety render it an ideal drug in acute cardiac emergencies, in marked congestive failure where oral digitalis is absorbed with some uncertainty and in those instances where one wishes to try another drug when digitalis fails. Gold[96] presented evidence which leaves no escape from the conclusion that amino phylline, theocalcin, theobromine with sodium salicylate, or any of the other xanthine compounds exert no action which is useful for the routine treatment of cardiac pain or myocardial infarction. Fineberg[97] reported the case of a patient with cardiac failure who received 343 injections of salyrgan and mercupurin over a period of seven and one-half years without any deleterious effect. Herrmann[98] stated that mercupurin seems to be the most efficient diuretic and at the same time the safest, as he had no evidence of unfavorable reaction in over 400 intravenous injections; and the new salyrgan-theophylline promises to be a similarly and most effective anti-edemic preparation. Volini and Levitt[99] found that the immediate effect of mercurial diuresis (and also of copious spontaneous diuresis) on the venous blood pressure is to produce a decrease; and this fall in venous pressure is an objective measure of clinical improvement in the treatment by mercurial diuretics of patients with heart failure and edema.

Angina Pectoris: Groedel[100] stated that ischemia or anoxemia of the myocardium or parts of it may be caused by an acute occlusion; by temporary or chronic impairment of the blood flow in sclerosed coronary vessels, or, due to overexertion, in healthy as well as in diseased coronaries; by abnormal vagal stimulation, for instance, during sleep; by an abnormal transient depression or elevation of blood pressure; by spasm of the coronary vessels under the momentary or prolonged influence of poisons like nicotine; by any other factor capable of producing spasm; and by a reflex starting in a diseased organ other than the heart. A study was made of the effect of 16 drugs, including a placebo—milk sugar—on angina pectoris due to coronary artery disease by Master, Jaffe and Dack;[101] the drugs included several xanthine derivatives, alcohol, phenobarbital, chloral, the bromides, the nitrites, a tissue extract, digitalis and two narcotics. No drug was found to exert any specific effect on the anginal syndrome, for the best results were obtained with a placebo, and the number of patients improved

ranged between 15% and 30% for all drugs. Massel[102] found that the xanthines offer a better chance for the relief of angina, in the ratio of 2:1, than aspirin, sodium bicarbonate, phenobarbital, or a combination of the latter two; that the combination of a xanthine with phenobarbital offer a much better chance for relief, in the ratio of more than 3:1, than the phenobarbital alone or in combination with sodium bicarbonate; and that the combination of a xanthine with phenobarbital offers a better chance for relief than a xanthine alone. From this he concluded that the xanthine derivatives do have a beneficial effect on the subjective state of the patient with angina pectoris and that this effect is definitely enhanced when the xanthine is combined with a sedative-like phenobarbital. Campbell[103] reported the case of a man who developed angina pectoris following a crushing accident. According to Raab and Schonbrunner[104] it has been suggested that in the so-called simple angina pectoris which appears during exertion, excitement, etc., the excessive elimination of epinephrine from the adrenals is concerned in the causation. Since it has been demonstrated that roentgen irradiation of the adrenals restricts the production of epinephrine, they tried this treatment on 38 patients with simple angina pectoris and found that 28 of them were either improved or entirely free from symptoms after one or several series of roentgen irradiations.

Cardiac Neurosis: Schnur[105] contends that cardiac neurosis is a distinct entity with characteristic findings which are recognizable even in the presence of organic heart disease. The criteria for a diagnosis of this condition are an inherited or acquired predisposition to neurosis; a definite precipitating factor; symptoms such as inframammary pain, weakness, sighing respiration, insomnia, dizziness, ringing in the ears, nervousness and irritability; inframammary tenderness and hyperalgesia and relief by simple procedures such as the therapeutic test described, namely, intradermal injection of small quantities of 2% novocain together with suitable suggestion, the latter being the more important factor.

Paroxysmal Tachycardia, Etc.: Campbell and Elliott[106] consider paroxysmal tachycardia a symptom rather than a disease. In a minority of patients it accompanies serious heart disease and the prognosis is grave; such cases are nearly always under observation for their heart disease before the onset of symptoms. In most patients it is not in itself of any grave significance, and is due to reflex causes more often than to any primary change in the heart muscle; the prognosis as regards life is excellent, unless it is of the rare ventricular type, unless appearing late in life, or unless before, there is already serious heart disease. Leibovici[107] reported the case of a man aged 29 who had developed a severe attack of paroxysmal tachycardia after an appendectomy; it was treated by the successful procainization of the left stellate ganglion with 20 cc. of a 1:200 solution. Cowan[108] stated that the alterations in the rhythm of the heart, as isolated signs, are not necessarily of serious significance. The occurrence of extrasystoles, per se, has not any sinister significance. It is true that if they recur rapidly for long periods, the mere rapidity of the cardiac contractions may produce cardiac failure, but, in the absence of signs of cardiac disease, any cardiac symptoms pass as soon as the normal rhythm is restored. Lyon and Rauh[109] examined 2,672 children with normal hearts and found extrasystoles in 59 (2.2%); and of 468 children with cardiac lesions observed the irregularity in 20 (4.3%). There was no evidence that the presence of extrasystoles by themselves, either caused any impairment of cardiac function or retarded the growth and development of the child.

Auricular Fibrillation: Smith and Boland[110] feel that quinidine is especially indicated when the patients are young persons who have idiopathic auricular fibrillation but who do not have evidence of heart disease; patients for whom it is most dangerous to administer quinidine are those who are elderly, who have rather serious hypertension and coronary sclerosis with marked cardiac enlargement, and who have had auricular fibrillation a long time. Burch[111] reported the case of a 61-year-old male with definite auricular fibrillation of 22 months' duration, in which the cardiac mechanism returned to normal without the aid of quinidine or related drugs. Messeloff[112] made observations on the use of quinidine sulfate in children and concluded that they tolerate large doses of the drug, as the usual adult dose may be given to them. Sokolow[113] gave quinidine sulfate to a middle-aged man with "benign" auricular fibrillation of uncertain etiology whose repeated emboli were the only manifestations of the disease; the result was satisfactory, with

restoration of sinus rhythm, subsidence of embolic phenomena, and maintenance of cardiac reserve. Gold et al.[114] stated that in digitalized patients with auricular fibrillation, the ventricle is maintained at a slow rate usually by the summation of a vagal and an extravagal factor; and, contrary to statements found in the literature, the results show that is not a matter of individual peculiarity, degree of heart failure or length of time the heart has been under digitalis.

Heart-Block: Aisner and Dorsey[115] reported a case of complete heart block resulting from overdosage with thyroid extract in an uncontrolled patient with obesity. Bishop and Carden[116] emphasized that bundle branch block is not in itself indicative of severe heart disease nor of a poor prognosis, for the character and degree of attended heart disease are the principal factors in determining the prognosis. Freund and Sokolov[117] pointed out that there is no "clinical picture" of bundle branch block. It is most frequently found in conjunction with definite heart disease; consequently the prognosis is at best no better than it is in cases of any form of myocardial disease. The most important factors to be considered are the general condition of the patient and the physical signs of cardiac damage. Patients who are in good condition and show little or no signs of cardiac embarrassment are not as a rule in immediate danger. Kaplan and Katz[118] made a follow-up study of a series of 126 patients whose electrocardiograms conformed to the criteria for various varieties of intraventricular block, all showing a QRS duration of 0.11 second or more. They also found that the character of the underlying disease, rather than the presence or absence of intraventricular block, determines the prognosis.

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PROGRESSIVE MEDICAL EDUCATION

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In celebrating this, the Centennial meeting of the Illinois State Medical Society, we reverently pay tribute to the men who fostered this organization. We cheerfully acknowledge the practices upon which this organization was founded and the principles for which these men stood. We pay deserving tribute to our past presidents and to the other officers who have guided the activities of this Society. They have protected, through legislation and improvement in education, those in the profession who follow in their wake.

The State of Illinois, admitted to the Union in 1818, had its first organized Medical Society in 1840. Superstition, inadequately interpreted observations, geographical hazards and phobias reacted adversely and heavily upon the Society so that development and progress were slow. Eventually the conditions began to improve and the "organization" idea began to spread as the fear of cliques was progressively broken down.

In 1843 Rush Medical School was opened, and in 1859 the Chicago Medical College began instruction in undergraduate training, the first such teaching institution to require a graded curriculum. It is a far cry from 1840 to 1940, so far as medical education is concerned. It would be impossible to adequately summarize the progress made, although the advances have been a challenge to all to keep pace. Imagine the undergraduate work of 1843. Imagine the graduate in 1846. Were even these three years required to complete his medical studies? History advises us that it was not. In fact, anyone desiring to become a physician had only to find a practitioner or "preceptor" with whom he could "flunky" for a period of a year and then put out his own shingle. In 1940 the picture is entirely different. In addition to four years of high school, the candidate for an M.D. degree, must have at least two years of premedical training, four years of medicine and at least one year of immediate graduate study. More than this, immediate graduate training has attained a high degree of perfection with residences, fellowships and associate instructorships being offered. In fact, medicine in general has been criticized because such training necessitates an expenditure of too much money to further these advances. Medical schools, too, have been criticized for so-called excessive expenditures in research. No! I do not think we have been extravagant We have, on the contrary, been well repaid for our efforts. We note with pride that the undergraduate and immediate graduate phase of medical education has reached an exceptional excellence in the United States. These criticisms are unjust when one reflects upon the need for training and research if medicine is to progress and if the patient is to receive the benefits of progressive medicine. One need only remember that the life span has been prolonged twenty-five years since 1840 by this process.

But what has been done with postgraduate education in this 100 years just passed? A justifiable criticism has come from the profession, as well as the laity, that postgraduate opportunities have not kept pace with undergraduate requirements. Why does this condition exist? Because postgraduate facilities have failed to reach the practitioner as well as undergraduate requirements have reached the student. Men who practice in hamlets, those upon whom the majority of our commonwealth must depend, cannot, for economic or geographic reasons, return to teaching centers for postgraduate work. Another reason for the failure to reach all is the highbrow boast that we, as physicians, are not interested in medical politics. We have been especially busy during the years from 1929 trying to make a living, interesting ourselves in

Chairman's address read before Section on Medicine of Illinois State Medical Society, May 22, 1940, Peoria.

the economic side of medicine at the expense of real scientific advancement.

In recognition of this, the medical schools are offering courses for practitioners in practically every subject in the undergraduate curriculum. The State Societies have made cooperative endeavors to provide refresher courses. In addition, many other facilities, such as a speakers' bureau, have been set up to provide the component County Societies with adequate programs. The annual state meeting provides another means of postgraduate instruction.

Have we utilized all these facilities to the fullest extent, and have we given the membership throughout the state the best we have to offer in postgraduate study? To do this we must have the complete cooperation of the individual practitioner. He must attend the meetings, he must take active part in Symposia and discussions and make himself a part of the postgraduate program. Physicians must not only get educated but must keep educated, for a license to practice medicine is a permit to study. Then it becomes the direct responsibility of the Medical Societies to stimulate the physician and help him to attain this achievement.

Have the Medical Societies assumed this responsibility? Yes! By Symposia on various subjects in order to crystallize medical information. Improvement has been made in the reading of papers. Joint sessions have been arranged to connect more clearly clinical manifestations. Orations in medicine and surgery are provided. Methods are selected for imparting informative data by consulting with participants on the program and having them consult with one another before papers are presented.

I would suggest that we further these activities by seminars, with emphasis on clinical manifestations; by pathological conferences, which include medical, surgical, laboratory and x-ray interpretation correlated to clinical findings. That programs be planned for the general man, who, after all, constitutes the overwhelming majority. These programs should not pattern too closely after the A.M.A. programs, for they are too highly specialized into Sections. Perhaps adoption by the State Society of such policies as are utilized for education by the states of Michigan, Idaho, New York and Indiana would be advantageous. The purpose of all medical conventions should be essentially that of postgraduate instruction. The program should be generally interesting, provide informative data, contribute something educational and should not be all work and no play but more work than play.

Too much emphasis cannot be placed on postgraduate education, and one of the chief responsibilities of the State Society is to further sponsor, through education, advertisement and even by insistence, more postgraduate work for and by the physicians of the state. It is axiomatic to any organization that attendance, morale and membership improve when Society meetings seem profitable.

418 Unity Building.

LABORATORY AIDS IN THE DIFFERENTIAL DIAGNOSIS OF COMA

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In the days when the treatment of coma had little to offer beyond nursing and hoping, a diagnosis was desirable but not of particular value and seldom urgently needed. Developments and improvements in therapeutics and progress in laboratory procedures give a high importance to early and accurate diagnosis. More particularly in those cases where a clinical history cannot be obtained an immediate diagnosis may make the difference between survival and death of the patient. Frequently, the laboratory can point directly to a diagnosis, notably in coma arising from some exogenous or endogenous poison. Even when the cause of coma is known laboratory examination often offers a means of estimating the degree of intoxication and the effect of treatment.

Coma may be defined in the broad sense as a loss of consciousness more or less profound and transient or prolonged, excepting the physiological condition of natural sleep. A list of conditions in which coma may occur early or in the later phases follows:

1. Cerebral anemia—a fundamental cause of sudden coma.

2. Mechanical injury of the brain—even slight concussion may result in loss of consciousness.

3. Convulsive attacks—these include epileptic seizures and many other causes of fits.

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4. Cerebral vascular attacks—include hemorrhage, thrombosis, and embolism.
5. Poisons:
   A. Exogenous agents; inhaled, ingested, or injected; carbon monoxide, alcohol, mercury bichloride and mercurials.
   B. Endogenous toxic products:
      a. toxemias associated with acute infections, typhoid fever, diphtheria.
      b. disturbed metabolism, diabetes mellitus, uremia, eclampsia, hypoglycemia, cholemia.
6. Local infection of brain and meninges.
7. Heat stroke.
8. Hysteria.

Laboratory tests are of greatest value in coma arising from causes grouped under poisons. However, available data indicate that coma most frequently results from such causative agents. Statistical studies on the incidence of coma in patients brought to hospitals have received little attention in the literature. Table 1 shows some of the data presented by Solomon and Aring[1] for the year 1933.

TABLE 1. CAUSES OF COMA AND THEIR MORTALITY

Disease or Poison	No. of Cases	Per Cent. of Total Comas	No. of Fatal Cases	Per Cent. Ending Fatally
Alcohol	690	59.1	14	2.0
Trauma	152	13.	48	31.5
Cardiac Decompensation	17	1.4	12	70.6
Cerebral Vascular Lesions	118	10.1	91	77.1
Poisoning	33	2.8	3	9.0
Epilepsy	28	2.4	0	0.0
Diabetes	20	1.7	11	55.0
Meningitis	20	1.7	20	100.0
Pneumonia	20	1.7	18	90.0
Exsanguination	10	0.9	10	100.0
C. N. S. Lues	7	0.6	0	0
Uremia	7	0.6	7	100.0
Eclampsia	7	0.6	3	42.8
Miscellaneous	38	3.2	26	68.4
Total	1,167	100.0	263	22.5%

The extent of laboratory aid obviously depends upon the facilities and personnel available. Where a chemist or toxicologist is at hand a considerable variety of tests may be carried out. However, with the general improvement in training of hospital laboratory technicians much may be expected with respect to a number of the simpler procedures. Recognizing the demand and need for descriptions of methods of detection of the more common poisons several of the most recently published clinical laboratory manuals have included the most trustworthy tests. Some of these are indicated below.

ALCOHOLISM

The usual toxic agent implied in the term "alcoholism" is ethyl alcohol though other alcohols may lead to coma. These include methyl alcohol and its commercial products such as antifreeze mixtures, denatured alcohol, and various lotions, amyl alcohol, prestone, and others. The clinical signs of alcoholism are well known: chemical examinations are of value in establishing the degree of intoxication and indicating whether or not alcoholism could account for all of the clinical symptoms in view of the possibility, and probability, of trauma associated with intoxication. One of the best criteria of alcoholic intoxication is the alcohol content of the blood because of the close parallel of this value with detrimental effects noted in carefully conducted experiments. Ten or a dozen methods have been described[2] for determining the concentration of alcohol in blood, and also in urine, saliva, cerebrospinal fluid, or breath. The amount of material required may be as small as 0.1 cc. The time for analysis varies from ten minutes to two hours. For most reliable results all the procedures must be carried out by a trained chemist or technician. The accumulation of data has established a definite, if not exact, relationship between blood or urine alcohol and degree of intoxication. Such correlation is shown in table 2 as prepared by Muehlberger.[3]

TABLE 2. STAGES OF INTOXICATION

Stage	Alcohol Concentration Blood	Urine	Clinical
Subclinical	0.01-0.12	0.01-0.16	Normal by ordinary observation, slight change by special tests.
Stimulation	0.09-0.21	0.13-0.29	Decreased inhibitions. Emotional instability. Slight incoordination slowing of stimuli response.
Confusion	0.18-0.30	0.26-0.42	Disturbance of sensation. Decrease pain sense. Staggering gait, slurred speech.
Stupor	0.27-0.39	0.38-0.54	Marked stimuli decrease. Approaching paralysis.
Coma	0.36-0.48	0.51-0.67	Complete unconsciousness. Depressed reflexes. Subnormal temperature. Impairment of circulation. Possible death.

TRAUMA AND CEREBRAL VASCULAR LESIONS

Among the traumatic comas, injuries to the head are the most common cause. Hence in such instances or when a cerebral vascular attack has taken place examination of the cerebrospinal fluid may be of some assistance. The presence

of a yellowish color (xanthochromic fluid) indicates that hemorrhage has previously occurred in some part of the central nervous system. However, a deep yellow fluid containing a large amount of protein points to spinal canal block. If red blood cells are absent the hemorrhage has stopped: blood in all specimens of spinal fluid collected indicates a recent hemorrhage as occurs in subarachnoid or intraventricular bleeding. The cell count, total protein estimation, and bacteriological examination are frequently helpful.

POISONS

The large number of poisons of exogenous origin that may cause coma contributes to the difficulty of laboratory tests and at the same time enhances the importance of chemical tests in arriving at the proper diagnosis. Clues to the type of toxic agent implicated in acute poisoning (other than alcoholic) may be given by the symptoms. Chemical tests for the detection of certain poisons have been described as follows:

Barbiturates—Cobalt color reaction;[4, 5] this test is not specific for the barbiturates but has proved to be of definite diagnostic value in comatose individuals. Delmonico[5] has described the clinical application of the following test:

25 cc. of urine are acidified with dilute (10 per cent.) sulphuric acid and extracted with an equal volume of chloroform. The chloroform extract is decolorized by means of charcoal, concentrated to a small volume (5 cc.). To 2 cc. of the extract are added 0.6 cc. of isopropylamine reagent (5 per cent. in absolute methyl alcohol) and 0.1 cc. of the cobaltous acetate reagent (1 per cent. in absolute methyl alcohol). In the presence of barbiturates a reddish- violet color appears immediately.

Cyanides—The characteristic odor of hydrocyanic acid may be detected in the breath; a very sensitive test that is applied to gastric or intestinal contents or blood consists in acidifying the material in a flask closed with a tightly fitting stopper into which has been inserted a strip of filter paper moistened in turn with a ten per cent. solution of guaiac or benzidine in alcohol and a 0.1 per cent. solution of copper sulphate in water. When the contents of the flask are gently warmed a blue or blue-green color develops on the paper strip in the presence of hydrocyanic acid or cyanides.

Bromides—A rapid qualitative test can be applied to urine; the specimen is decolorized with charcoal, acidified with acetic acid, and a small amount of 0.5 per cent. solution of gold chloride in water added. Development of a brown color indicates the presence of bromides. Quantitative estimation of the blood concentration may be carried out by a somewhat similar technique but is unnecessary should a negative test be obtained with the urine. Blood bromide concentrations of 200-300 mg. per cent. and higher are to be expected.

Metals—The group of metallic poisons most frequently encountered are mercury, arsenic, phosphorus, barium, lead and iodine. The well-known Reinsch test applied to gastric or intestinal contents serves as a short, ready, and sensitive test for the detection of mercury, arsenic, antimony and bismuth. Gettler[6] has described a modification of this test as applied to urine.

Iodine is its own indicator both by virtue of its own deep brown color and the blue color that it gives with a suspension of starch. Phosphorus, barium, and lead are more difficult to identify; but, fortunately, are rarely responsible for coma.

Benzene, nitrobenzene, and derivatives—Volatile organic poisons are largely eliminated through the lungs; a small amount is oxidized and excreted in the urine in the form of phenol and conjugated sulphates. Yant and co-workers[9] have developed a microcolorimetric method for the determination of benzene and also[10] have recommended the determination of the ratio of inorganic to organic sulfates in the urine as an index of benzene intoxication.

Aniline, acetanilid, and phenacetin—The indophenol reaction applied to the urine is positive in the presence of compounds containing a free primary aromatic amino group; hence it is necessary first to hydrolyse by boiling the urine with acid before applying the test. This test is time-consuming but presents no special difficulties. Positive results ordinarily are obtained only after fairly large doses have been taken.

Carbon monoxide—The clinical signs of carbon monoxide poisoning have long been known, and for some time there have been available compact and convenient sets that permit rapid estimation of the blood carbon monoxide concentration to within ten per cent. of the true value. For concentrations lower than about 20 per cent. the gasometric method of Van Slyke and Salvesen[7] using either the volumetric or

manometric apparatus is the most reliable. The gasometric procedure requires special apparatus and a properly trained technician.

Alkaloids—Of the alkaloidal poisons morphine, opium, codeine, heroin, and strychnine are most frequently responsible for coma. Contraction of the pupil of the eye and a slow respiratory rate are distinctive symptoms for opium and the opiates. For chemical tests the contents of the stomach or the urine serve as tests materials. The usual extraction procedure for alkaloids is followed and either Frohde's or Marquis' test applied. Strychnine is readily detected in the purified residue by its bitter taste and the fading purple test. A biologic test using a frog is capable of detecting very small amount of strychnine.

The relative importance of toxic agents causing death as reported by Alexander, Moore, and Leary[8] for the ten-year period 1928 through 1937 for the State of Massachusetts is shown in table 3.

TABLE 3. RELATIVE IMPORTANCE OF VARIOUS TOXIC SUBSTANCES CAUSING DEATH IN 8,661 CASES OCCURRING DURING 1928-1937

	No. of Deaths	Per Cent. of Total Deaths	Per Cent. of Poisons
Alcohol	4,752	5.38	52
Illuminating Gas	1,830	2.08	21
Non-Therapeutic Chemicals	918	1.04	11
Carbon Monoxide	690	0.79	8
Surgical Anesthetics	345	0.39	4
Sedative Drugs	271	0.31	3
Non-Sedative Drugs	102	0.12	1
	8,908	10.11	100

Laboratory procedures that may be applied to patients in coma arising from endogenous toxic products are indicated in Table 4. The diagnosis of diabetic coma does not rest on glycosuria alone. There should be found in the urine strongly positive tests for the ketone bodies, acetone and diacetic acid, and large amounts of glucose. Determination of the carbon dioxide capacity or content of the plasma gives a measure of the degree of acidosis that accompanies diabetic coma and nephritis. Either value may be obtained within 15 minutes after receipt of the blood specimen using the Van Slyke gasometric apparatus. Blood for carbon dioxide content must be collected under oil and is therefore a little more inconvenient than the CO_2 capacity. However, the former is the more desirable value, particularly in children because it gives the actual content of CO_2 in the circulating blood while the latter may vary with the cell volume and cause misconception in anemia and other conditions.

Uremia has been called the great imitator among diseases; it has often been the diagnosis when it did not exist, and frequently remained undiscovered or not diagnosed when present. In this connection it should be noted that albumin and casts in the urine are not sufficient evidence of renal damage, and, further, that coma in acute nephritis is not necessarily caused by uremia. A urinary odor to the breath is significant. A diagnosis of uremia is substantiated by values of 120 mg. per cent. or higher for blood non-protein nitrogen or by concentrations of 80 mg. per cent. or higher for blood urea nitrogen.

In preeclampsia and eclampsia the total non-protein nitrogen and urea nitrogen are usually within normal limits. An increase in blood uric acid is the most common observation; indeed, frequently it is the only demonstrable abnormality in the blood constituents. When eclampsia is suspected both blood uric acid and urea nitrogen or nonprotein nitrogen should be determined.

TABLE 4. LABORATORY TESTS IN DIFFERENTIAL DIAGNOSIS OF COMA CAUSED BY VARIOUS CONDITIONS

	Urine		Blood		Plasma
	Glucose	Acetone	Glucose	Urea	CO_2 Capacity
Diabetes	marked	present	high	normal or high	low
Uremia	usually present	present	normal usually	high	normal or low
Insulin Reaction	absent after initial voiding	may be present	low	normal	normal
Meningitis	present	.....	normal or sl. increase	normal	normal
Brain Tumor	absent	.....	normal or sl. increase	normal	normal
Cerebral Hemorrhage	usually present	.....	normal or sl. increase	normal	normal

SUMMARY

Laboratory examinations that lend themselves to the differential diagnosis of coma have been

described. Details of some of the simpler tests are given.

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ACUTE GANGRENOUS APPENDICITIS ASSOCIATED WITH EMBOLIC OCCLUSION OF THE CORONARY ARTERY

Case Report

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CHICAGO

Coronary occlusion following surgical intervention for an abdominal condition has been noted with increasing frequency since the reports of Saphir, Priest, Hamburger and Katz;[1] Campbell[2] and Master, Dack and Jaffe.[3] However, we have failed to find any reports of coronary occlusion occurring during the course of an acute abdominal condition such as our case. Because of the rarity of coronary embolism occurring in the presence of a gangrenous appendix, we feel that the case is of unusual interest to warrant its report.

In a review of 12,000 consecutive necropsies performed at the Cook County Hospital from 1929 to July, 1939, inclusive, the association of embolic occlusion of the coronary artery and acute gangrenous appendicitis has been observed in only one case, the one to be reported. There were 120 cases of fatal appendicitis, 48 were operated upon and 72 were not.

REPORT OF CASE

History:

W. S., a 50-year old white male was admitted to the surgical service of the Cook County Hospital on August 12, 1938, complaining of severe abdominal pain. The onset of symptoms dated back to August 6th, when he was seized with periumbilical pain which persisted until his admission. The pain was severe enough to make him miserable. He ate nothing because of the pain and vomited several times each day. His bowel movements were watery in nature. The morning of admission he was suddenly seized with very severe lower abdominal pain radiating from the umbilical to the pubic area.

Physical Examination:

Revealed a white male lying in bed who appeared acutely ill. The heart examination was essentially negative and the lungs were clear and resonant. The abdomen was flat and markedly tender throughout, tenderness being more marked in the right lower quadrant. The abdominal wall was rigid, the obturator sign was positive, the psoas and Rovsing signs were negative. There was marked rebound tenderness, the Murphy Punch was one plus on the right side and the peristaltic sounds were absent.

Clinical Impression:

Ruptured appendix with generalized peritonitis.

Laboratory:

The blood count on admission revealed a white count of 4,400 which rose the following day to 9,400. Fluoroscopic examination revealed no free air in the abdomen. The patient felt somewhat better the next day, but continued to have cramp-like pains in the right lower quadrant.

Course:

He was placed on conservative management and on August 15th, as mass was felt in the right lower quadrant. His temperature ran a septic course and he was given intravenous fluids. A Levine tube was inserted into the stomach and he was kept in Fowler's position. On August 18th, he complained of pain in his left leg and knee which had become swollen. He became cyanotic, his respirations were labored and he appeared terminal. A diagnosis of pulmonary embolism was made and he expired August 18th, six days after admission or two weeks after the onset of his illness.

Necropsy:—(Performed by J. D. Kirshbaum, M. D.)

The body was well nourished. The abdominal cavity contained 200 cc. of a cloudy yellowish fluid. The serosa of the intestines was dull, injected and loops of bowels were loosely adherent to the anterior abdominal wall. The base of the appendix had sloughed off, leaving an opening in the cecum. The appendix, itself, was friable and discolored a purple green. The heart was enlarged and weighed 490 grams. The myocardium was soft, friable, and light brown in color. The wall of the left ventricle measured 14 mm. and the right ventricle measured 4 mm. The cardiac chambers, especially the ventricles, were dilated. The endocardium of the left ventricle was thickened especially in the region of the apex. Between the trabeculae carnae near the apex of the left ventricle, there was a firmly adherent

From the Department of Pathology, Cook County Hospital, Chicago.

dark pulple red blood clot measuring 5x2.5 cms. The aorta measured 77 mm. in circumference, and the intima was smooth. The orifice of the descending branch of the left coronary artery was occluded by a soft purple red blood clot which extended downwards for 22 mm. into the lumen of the artery (fig. 1). The intima in the region of the clot was roughened by hyaline and fatty plaques.

The lungs were distended and the posterior portions were firm non-crepitant. On sectioning, the upper and middle lobes were light purple red and moist. The lower lobes were darker purple red and moist with blood. The main branches of the pulmonary artery were occluded by soft, loosely adherent dark

Figure 1. Photograph of the heart showing the embolus occluding the left coronary orifice with extension into the descending branch. Not large mural thrombus in the left ventricle.

purple-gray blood clots. The branches of the left pulmonary artery were also occluded by similar clots. The mucosa of the bronchi were injected.

Anatomic Diagnosis:

Acute gangreous appendicitis with perforation and sequestration; diffuse suppurative peritonitis; embolic occlusion of the descending branch of the left coronary artery; embolic occlusion of the main branches of the pulmonary artery; mural thrombus in the left ventricle and thrombosis of the left iliac and femoral veins.

DISCUSSION

The course of events in our case may be summarized as follows: The patient had an essential hypertension and developed an acute appendicitis. The pathological changes in the appendix progressed to gangrene, perforation, sequestration and a diffuse peritonitis. In the presence of the peritonitis the heart decompensated, mural thrombi developed in the left ventricle, a piece broke off and occluded the orifice of the left coronary artery. The stasis of the blood flow as a result of the failing heart and the infectious process in the abdomen predisposed to the throbus formation in the left iliac vein which was the source for the pulmonary embolism, the latter being the immediate cause of death.

That a patient past middle life suffering from an acute appendicitis may also be inflicted with a second pathological process should be kept in mind. This case also emphasizes the importance of a careful heart examination and blood pressure reading in every patient in whom surgical interference is anticipated.

CONCLUSION

A case of gangrenous appendicitis with perforation and diffuse peritonitis complicated by an embolic occlusion of the coronary artery is here presented.

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FOREIGN BODIES IN THE VERMIFORM APPENDIX, WITH REPORT OF CASE

MAX C. EHRLICH, B. S., M. D., F. A. C. S.
CHICAGO

Foreign bodies in the appendix have been described for more than two centuries. In the early reports they were described as incidental findings at postmorten examinations. Mitchell[1] reports the earliest probable case described by Ruysch in 1691. Hevin[2] in 1743 found at postmortem, shot in large quantities in the appendices of persons who had eaten a great deal of game. These cases were apparently symptomless.

The first authentic case reported was that of a pin in the appendix, that of Mestivier[3] of Bordeaux in 1757, also reported by Mitchell. Mestivier reported the case of a man who developed a large abscess in the right side of his abdomen. At autopsy an eroded pin was found in the lumen of the appendix. This first definite

*From Wesley Memorial Hospital.

case report led to the possible relationship between foreign bodies and inflammation of the appendix. Somewhat later it was thought, that foreign bodies were always the cause of appendicitis. However, further investigation has shown that foreign bodies are only occasionally responsible for acute appendicitis. Actually, *true foreign bodies in the appendix* are of rare occurrence as shown by a number of observers. Fowler,[4] in a review of 10,095 cases of appendicitis, reported by 17 observers, found foreign bodies in 2.6 per cent. of cases. Mitchell[1] reports foreign bodies in 7 per cent. Fowler[4] found three cases of foreign bodies in 2,000 cases. DeForest[5] did not find a single case of foreign body appendicitis in several thousand cases. Deaver[6] states, in regard to foreign bodies in appendicitis, "they are very rare as an exciting cause." Osler and McCrae[7] agree with the statement of Deaver. Kelly[8] found four instances of foreign bodies in 1,000 cases. Bell[9] found five instances of foreign bodies in 1,000 cases. Many instances of fecal concretions have been mistaken for foreign bodies from their similarity in size, shape and appearance, but careful examination of these reveals their true identity.

Parasites and worms, especially oxyuris, are far too common to warrant other than passing mention. Gordon[10] has shown, in a study of 71 appendices showing inflammation and containing oxyuris, that in none of them could the inflammation be attributed to the parasites, since the worms were present only in the lumen of the appendix, with no evidence of mucosal involvement. He concludes that oxyuris vermicularis is not a cause of appendiceal pathologic changes.

Among the large variety of foreign bodies that have been found in the appendix, are included shot, pins, needles, nails, bullets, stones, pebbles, screws, buttons, seeds, pieces of bone, pieces of wood, iron filings, beans, oat-hulls, cherry stones, chestnuts, bristles, egg-shell, hair, teeth, globules of solder, vertebra of fish, portions of porcelain, portions of darning needles and hat-pins, twigs and straw. Of these many types of foreign bodies, common pins were most frequently encountered. Mahoney[11] found 40 common pins among 74 cases of foreign body appendicitis. Of 110 foreign bodies reported by Berger,[12] 19 were pins. Berthelier[13] found seven pins in 29 cases of foreign body appendicitis. The least common foreign bodies found in the appendix were teeth. Only three cases have thus far been reported in the literature, one by Upton,[14] one by Christeller and Mayer,[15] and one by Meyer and Rosi.[16] Most cases of pins in the appendix were found in men, not women, and in children under ten years of age. Strangely enough, Kelly[8] found only two cases in seamstresses. The mortality of cases of pins in the appendix is very high—44 to 55 per cent. according to Mahoney.[11]

CASE REPORT.—A female child, aged 2½ years, was admitted to the Wesley Memorial hospital several days after the onset of pain in the abdomen, which the child described as "my tummy hurts." During the two days before admission the child was nauseated but did not vomit. Food was refused most of the time and on the day of admission to the hospital the child had eaten nothing and had taken only small amounts of water.

Previous history revealed nothing of importance. Birth followed a moderately prolonged labor which terminated in an outlet forceps delivery. There were no illnesses previous to admission to the hospital except one common cold. Development of the child was normal and uneventful.

Physical examination revealed a well developed female child past two years of age. Rectal examination, mouth, throat, neck and chest showed no abnormalities. The abdomen was tender everywhere, but extremely tender in the right lower quadrant with marked rigidity on the right side of the lower abdomen. No masses were palpated. Extremities were negative and all reflexes were normal. Rectal temperature was 101.2 F. Pulse was 120, and respirations 28 per minute. Erythrocytes 5,120,000, Leucocytes 16,000 and hemoglobin 92 per cent. (Newcommer). Polymorphonuclear neutrophiles predominated in the differential blood count (86 per cent.). Urine analysis was normal. Diagnosis of acute appendicitis was made and the patient was operated upon within an hour after admission.

The abdomen was opened with a McBirney incision and the peritoneal cavity was found to contain a small amount of thin exudate which was odorless. The appendix was readily identified and delivered, no adhesions being present. The appendix did not appear to be acutely inflamed, but had considerable injection of the blood vessels. It measured about two inch in length. There was a sharp angulation of the middle portion of the appendix which has caused a shortening of about 1½ centimeters in the length. This angulation produced a bulbous formation, which later was found to contain a common pin it its lumen. Removal of the appendix was accomplished in the usual manner and the abdomen was closed in layers without drainage. Recovery was entirely uneventful, the highest temperature recorded being 101.8 F. per rectum, and the patient was discharged from the hospital on the seventh day.

Pathological Description (By Dr. E. R. Strauser, Pathologist, Wesley Memorial Hospital). Gross Findings:—The appendix measures 3½ x ½ x ¾ centimeters. A sharp compound angulation of the middle portion of the appendix has caused a shortening of

about 1½ centimeters in the length. Marked fibrosis about this overlapped portion of the appendix has greatly interfered with recognition of this marked angulation by gross examination. The serosa is slightly injected and dull in the distal portion of the appendix where the wall is irregularly thickened by fibrosis. The distal straight segment of the appendix tapers gradually to a small firm tip, distally. Underneath the serosa of the distal end of the appendix is the rusty head of a common pin, the shaft of the pin extending proximally through the lumen of the distal segment of the appendix, so that the tip rests in a mass of fibrotic tissue at the fold in the appendix. The mucosa about the pin is discolored and apparently replaced by fibrous tissue. There is little change in the proximal portion of the appendix, except for slight fibrous thickening of the wall (See fig. 1).

Microscopic Examination:—Examination of the sections taken from the angulated portion of the appendix, show marked fibrosis about the tip of the pin. Most of the mucosa and submucosa have been replaced by fibrous tissue in which there is a moderate infiltration of lymphocytes, large monocytes and eosinophiles. Neutrophiles are not very numerous. The serosa of this portion of the appendix is edematous and its blood vessels are congested with red cells of leucocytes. Other portions of the appendix are less involved. Pressure atrophy of the distal end of the appendix, produced by the head of the pin, has resulted in only the thin covering of the serosa over the pin head. The proximal portion of the appendix shows no significant changes (See fig. 2).

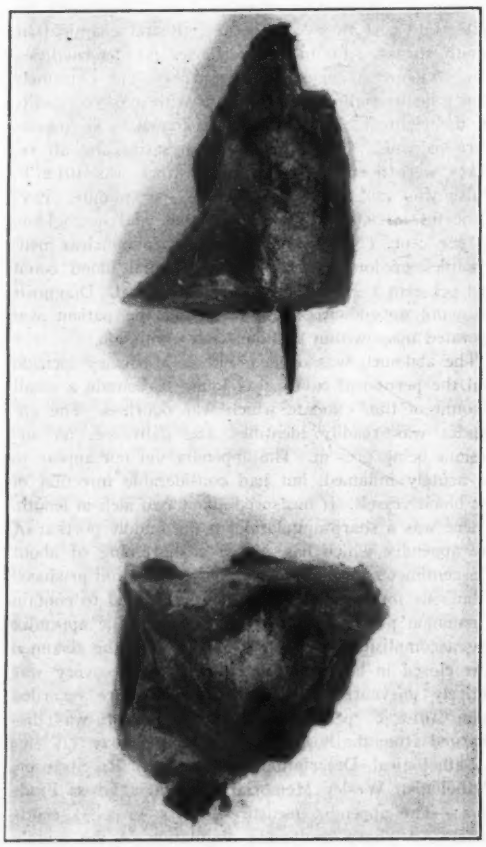

Figure 1

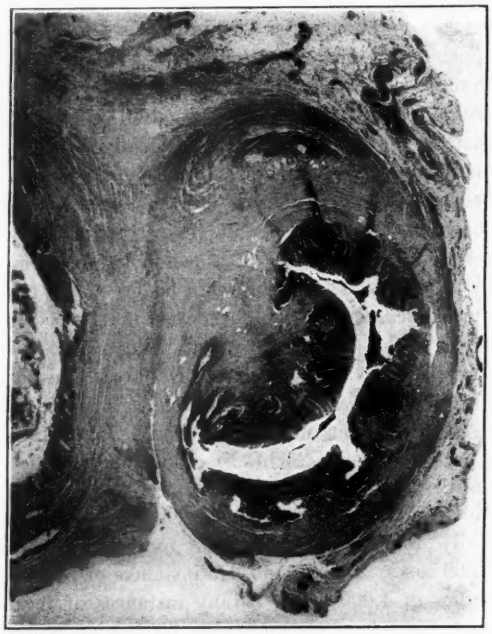

Figure 2

Diagnosis: Chronic inflammatory reaction about foreign body of appendix.

COMMENT

Pointed bodies in the appendix directly produce inflammation of the appendix, but in most instances, especially in the case of pins, there is evidence that the foreign body has been present for a long time before the acute process develops. In many cases the foreign body lay quiescent in the appendix until encrusted with calcareous material, and finally, determining the site of a perforation rather than inducing the acute inflammatory attack. Bodies of light weight like grape seeds and cherry stones do not often enter the lumen of the appendix. The explanation for this is that because of their shape and weight, pointed and heavy bodies more readily become engaged in the appendiceal orifice and pass into the canal. Sharp foreign bodies may cause

injury to the mucosa and immediately give rise to an acute process with occasional perforation. Foreign bodies are found eroded and encrusted after remaining for years in the appendix. Some foreign bodies remain dormant in the appendix for years, an acute attack being provoked by some form of trauma, such as a kick in the abdomen or a severe fall. In the majority of instances there is evidence that the foreign body excites a chronic inflammatory reaction which ultimately results in an acute process or leads to abscess formation, with peritonitis and sometimes resulting in death. Symptoms produced by a foreign body vary from mild attacks of pain to those of a most severe acute attack. Smooth, rounded objects may cause no injury to the mucosa, but may produce an obstruction of the lumen of the appendix with abscess formation. Constant irritation of the lumen of the appendix by a foreign body may produce varying degrees of chronic inflammation of the appendix. Metastatic abscesses of the liver are common in suppurative appendicitis from foreign bodies. Perforation of the appendix is frequent.

CONCLUSIONS AND SUMMARY

1. True foreign bodies in the appendix are of rare occurrence.

2. In the production of appendicitis, foreign bodies are very rare as an exciting cause.

3. Common pins are the most frequently encountered foreign bodies found in the appendix, and chiefly found in males.

4. Symptoms of appendicitis due to foreign bodies depend on injury to the mucosa, or from obstruction, partial or complete, to the lumen of the appendix.

5. Mortality of appendicitis due to pins is very high.

6. In the case here reported, there was not any knowledge of the pin having been swallowed.

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ARITHMETIC PROBLEM—If the average case of minimal tuberculosis admitted to the sanatorium stays 8 months, and the average case of moderately advanced tuberculosis stays 23 months, how much could be saved by early diagnosis?

In Connecticut where it costs about $1,000 to keep a patient in the state sanatorium for a year and where only 10% of the admissions are in the minimal stage, it is estimated as hundreds of thousands of dollars by the Connecticut State Tuberculosis Commission.

A RECENT STUDY OF 14 GENERAL HOSPITALS IN NEW YORK STATE where a routine chest X-ray was made on all cases admitted, showed 1.2% of 4,000 patients to have significant lesions and 0.7% of adult admissions were found to have hitherto unsuspected active tuberculosis. If the same condition prevails generally, it means that 45,000 unrecognized cases of tuberculosis were admitted to general hospitals during 1937. Whitney, J., Amer. Rev. of Tuber., Aug., 1939.

FAMOUS SAYINGS

Samson: "I'm strong for you, kid."
David: "The bigger they are, the harder they fall."
Nero: "Hot stuff! Keep the home fires burning."
Cleopatra: "You're an easy Mark Antony."
Helen: "So this is Paris."
Noah: "It floats."
Methuselah: "The first hundred years are the hardest."

On one point there is no longer debate. The need of medical and social supervision for arrested cases of tuberculosis over a prolonged period is clearly indicated. It is the only insurance against failure regardless of whatever procedure is adopted in the individual case.—Nat'l Tuber. Assn. Ann'l Rep., 1938.

In our town there is an old maid with a sour puss who, according to the local gossips, was advised by a nearby crack-pot quack to add saccharine to her douche.—*Submitted by a subscriber.*

THE CASE FOR PRIVATE PRACTICE*

The following article taken from *Nation's Business,* May, 1940, should open the eyes of the medical profession. It shows how far regimentation and dictocracy have progressed in their drive to absorb the science and art of medicine, and why it must be stopped. It is one of the most constructive articles on medical economics that has appeared in a lay journal. It is a "crushing" argument against Government controlled medicine.

It has been said that, wherever two or more persons meet and talk, it is a sporting bet that they will discuss one of three matters: sickness, death or the cost of things. Whether or not this is literally true, it scarcely exaggerates the human interest in those ills to which flesh is heir.

Credulity reaches its farthest limits of absurdity in these three subjects. It would be hard to imagine a fallacy with regard to any one of them so fantastic that at one time or another groups of people have not embraced it. Even in these enlightened times, thousands believe that washing the hands in stump water will kill warts, others that laying on of hands will make a paralytic take up his bed and walk, and still others that the same bottled elixir will "cure" hardening of the liver, prostatic enlargement and gallstones.

Every imaginable superstition clusters around death and its aftermath. And as for the price of things, no form of credulity is more current today than the belief that the prices of goods and services can be set by decree and in complete defiance of supply and demand.

Small wonder, then, that people listen to medical quacks and vote as political quacks prescribe for the royal road to health. A blind faith has grown up in the land that money buys health under any and all circumstances. If health can be bought at so much a head, then it follows that those who have must be made to disgorge so that others who have not may live. Ergo, the man who owns a home and a bank account is guilty of murder because babies die among families who have neither.

There often is some degree of correlation between certain diseases and economic status. Havelock Ellis showed that there is also a modicum of correlation between tuberculosis and genius. But to reason from his findings that people have tuberculosis because they are geniuses or that they are geniuses because they have tuberculosis, is as absurd as the current assumption that people who are sick are sick because they are poor or that, if they are economic failures, it is because they have been sick.

Yet writers in the scientific field, like the eminent Paul de Kruif, shout this fallacy in emotional, intemperate language. When he writes of "thousands dying simply because they hadn't the wherewithal to pay" for treatment, many of the intellectually defenseless believe him. When he scorches the paper in his type-

*This article is one of five of a series on "Free Enterprise" against "Dictocracy" published by *Nation's Business,* official organ of the United States Chamber of Commerce. It should prove an eye-opener to business men, big and little—those who will foot the gigantic bill, if any when the government takes over the job of running the healing arts.

A beginning must be made with the task of reconciling the laboring classes with the State. Whoever has a pension assured to him in his old age is much more contented and easy to manage than the man who has no such prospect. Compare a servant in a private house and one attached to a Government office or to the Court; the latter, because he looks forward to a pension, will put up with a great deal more.

—*Prince Otto Von Bismarck, the father of social insurance*

WHEN THE PEOPLE PRESCRIBE

The public inclination to follow medical Pied Pipers is shown in the case of John R. Brinkley, the "goat gland doctor." Deprived of his license to practice medicine in Kansas, Brinkley gained a great following with his radio station through which his voice wafted daily the promises of miraculous cures. He ran for Governor of Kansas, entering too late to get his name printed on the ballot, made a whirlwind radio campaign, and hourly "taught" his hearers how to spell his name. Some 25,000 adult citizens flunked their spelling examination on election day or Brinkley would have been Governor.

In Oklahoma, 20,000 voters wrote in his name on their ballots under the impression that he was a candidate in the Sooner State.

When his radio license was cancelled, Brinkley went to Mexico and acquired another station. He testified in court that his income in 1937 was $1,300,000.

It is conceivable that political boards in charge of medicine, although composed of men of courage and integrity, would have difficulty in resisting the cock-eyed demands of a patent medicine electorate.

—*From "Planning the Less Abundant Life"*

writer with flaming passages about "A future American citizenry rachitic, consumptive, undersized, sapped and degraded," sympathetic Americans, always moved through the heart more easily than the mind, mutter that we must do something about it.

One doesn't have to look far to see that this economic interpretation of the healthy life is grossly exaggerated. All the money of John D. Rockefeller could not buy the health enjoyed by the humblest "white wing" or stevedore. The young bruisers from well-to-do families who go to Harvard, Yale and Princeton have no corner on health, as their football record in recent years demonstrates when compared with that of Notre Dame, Southern Methodist or Minnesota. Joe Louis is not exactly a weakling, although he came from an underprivileged Alabama family.

"UNDERPRIVILEGED" CAN TAKE IT

An industrial psychologist at Harvard recently tested a group of young sharecroppers from Mississippi and an equal number of average Harvard boys to compare their resistance to fatigue. He found the sharecroppers superior to the "aristocrats" in capacity to undergo severe physical trials, although every one of them belonged to the "ill-clad, ill-housed, ill-fed and ill-doctored" bloc of the nation.

But these are isolated cases and not representative, you will be told. Let's look at the same question statistically. We find that the District of Columbia, seat of the national Government, has the highest death rate in the nation, if we except Arizona and New Mexico, both of which have a large percentage of tuberculous health seekers from the East among their population. The District also boasts a higher *per capita* income than any one of the 48 states. That is reverse correlation, but we will not ape the political health faddists by trying to reason from it that better incomes necessarily mean worse health. It can be pointed out, however, that the four states with the lowest death rates are North Dakota, South Dakota, Oklahoma and Arkansas. Three of these are definitely "have-not" states.

With all this credulous preoccupation with bodily ills and the plausibility of claims for their class origin, it is not surprising that those who seek ruling power in perpetuity should make health programs to advance their purposes. Such programs provide something else that government can do to win the gratitude of people too thoughtless to recognize Greeks when they come bearing gifts labeled "other people's money."

For centuries men fought to free their churches from state control. Eventually, after rivers of blood had been shed, they succeeded and now in large areas of the world we have religious freedom. During all those centuries, the state had nothing to do with the treatment of sickness. Now it is proposed to turn that function over to government, presumably to create a problem for posterity—the job of winning back freedom over their bodies as they once wrested freedom over the custody of their souls from government.

The most ambitious of health programs is compulsory health insurance. Like others, it is an importation from Europe.

ESCAPE FROM LIFE

And one of the things the citizen believes is that life is too difficult and severe; that there ought to be an easy way to solve every problem and to get the good things of the world without the sacrifice and self-denial which have usually been the price at which they were to be obtained. Therefore he likes to believe, when he is told it, that medical care ought to be provided by government at a cost of little or nothing to him.

The doctor looks at the citizen and finds him a willing tool in the hands of the professional political charlatan who continually tells him what he already likes to believe. It would be funny, were it not tragic, to trace the history of his befuddlement by one after another sweet singer of halcyon songs, who have played upon his weakness . . . why is it since these various panaceas to ease the lot of the citizen have failed, the citizen still seems eager to be fooled by each new plan? I believe the answer is because these plans offer the citizen an escape from the hard realities of life, just as phantasy does.

—Dr. Terry M. Townsend, President, Medical Society of the State of New York

In 1883, Bismarck proposed in Germany the first system of compulsory sickness insurance which was eventually to set the pattern for social security legislation in many nations. It was a system by which the employee contributes a portion of his wages, the employer another portion, and the state a third portion.

A deceptive device, since the worker pays most of the other two shares as well as his own. He helps to pays his employer's share, since the tax represents an added cost of doing business and must therefore be passed on to him and others wrapped up in the goods the employer makes and sells. And of course he pays the Government's share in the taxes also hidden in the goods he buys.

If the American worker were to have an actual estimation of his contribution to social security taxes by these three different routes, he would discover that the amount he pays against the hazards of old age and unemployment and as a contribution to other services under Title 6 of the Social Security Act represents a considerable sum.

Since the establishment in Germany of the concept that the State might begin to take over for the worker many of the responsibilities which American workers have always shouldered themselves, the political value of the system became apparent to political leaders in many another state. Gradually the scheme has spread, with variations and modifications, to other countries. It is safe to say, however, that, in not one of them, is there a system which provides a type of medical service even approximately as good as that available to the vast majority of American workers today. Increasingly in some nations more and more of the private functions of the individual were taken over. The German worker contributes a large part of his wages to the State so that the State may carry out for him many of the most intimate and personal functions of his life.

BRITISH SYSTEM CHILD OF POLITICS

In England after 1911, when it seemed possible that the Labor Party might take away from the then dominant Liberal Party the control of the Government, Lloyd George offered to British workers a similar system and combined with it the dole. That combination is probably subject even to more criticism than some of those which are being offered to Americans today. Under the British compulsory sickness insurance system, the worker receives nothing but a general practitioner's service, the effectiveness of which is open to question. When he requires the services of hospitals and specialists, he becomes essentially a charity patient. Unfortunately, however, as a result of this system, the voluntary hospitals of Great Britain which give this service find themselves facing a catastrophe—an end of their existence as independent institutions. Of course, it is being proposed that the Government take over the administration and support of these hospitals.

Now it must be remembered that, in the United States, we have developed what is probably the finest hospital system in the world. The majority of the hospitals which give medical care are established on a non-profit voluntary basis with the support of the Catholic, the Protestant, the Jewish and other churches, and of

SOVIET MEDICINE

We came, unluckily, to know a lot more about Soviet medical practice than most of our colleagues. Like the "stable" currency and the wonderful educational methods, the socialized medicine under its official statistical surface was a snarl of contradictions, shortages and ineptness. Doctors regarded their obligatory work for the state as an exaction and depended on private practice for their real income. The more famous medical specialists did not budge for less than 50 or 100 rubles; often it required "pull" to get their services at any price. The public health service was by all odds inferior to the free public and charitable health services available to the poor in cities like New York and Chicago.

—*Eugene Lyons in "Assignment in Utopia"*

trades, organizations, and communities. The care of the sick has been a fundamental spiritual motive in every great religion since the earliest times. The extent to which removal of this motive from religious groups would interfere seriously with their spiritual function is a matter which deserves the most careful thought and consideration.

The years from 1905 onward witnessed the development in the United States of a new profession—the social service group—today numbering many thousands of persons whose full time is spent in the administration, distribution and control of public and private assistance to the needy and sometimes to those not quite so needy. Increasingly, members of that group have taken their inspiration for future development from foreign patterns. They observe housing projects in Australia and Sweden and state administered services in Russia and Germany. They try to transplant similiar mechanisms into the American system of living.

They became more and more articulate in these attempts to change medical service at the time of the World War. During that conflict, mass handling of men began to be recognized as an exceedingly useful performance to achieve a definite objective. After the war, there was an effort to carry this mass handling of men over into civil life. All sorts of strange schemes were proposed for reorganizing medical service on a mass basis.

One of these was the Sheppard-Towner Act, passed shortly after the war, which provided federal aid for maternity care. Properly wrapped in Mother's Day sentiments, it appealed powerfully to the emerging social consciousness of that decade. It functioned only where the states voted their own enabling legislation. The decrease in infant mortality in the seven or eight years the act was in force was not so marked as it had been previously, and no better than it was afterward. Illinois and Massachusetts, the two states that did not accept the federal handouts, showed an improvement equal to those that did subjugate themselves before the growing federal colossus.

PATIENTS ARE PEOPLE, NOT ROBOTS

The physicians themselves had the concept that the finest professional service was rendered when there was free choice of doctor by the patient, mutual responsibility between patient and doctor, and the maintenance of a personal relationship between doctor and patient which considered the patient as a human being and not merely as an ailing unit in a great machine.

The economists, however, believed that the reduction of medical service costs made possible with mass handling might be sufficient reward for the destruction of these values. Here, there and everywhere proposals were made for the setting up of groups of doctors to administer service as units with complete changes in the method of payment for the service. Most articulate was the group who insisted that the state must take over medical service, administering it under a system of compulsory sickness insurance.

This the medical profession opposed particularly as representing waste through the expenditure of vast sums on governmental bureaus and institutions with all the officials and employees re-

JUST "SOCIAL" WAGES

Compulsory health insurance was instituted in Germany, where it first came into fashion, not as a means of bettering the German people's health or welfare, but as a donated dole in lieu of wages.

—*Dr. Samuel J. Kopetzky*

QUANTITY MEDICAL SERVICE

What is the general practitioner in England today but a glorified first-aid man? Quack patients beget quack doctors. It is a question, from the general practitioner's point of view, of getting as many patients as he can on his list and getting the consultations over as fast as he can. Only by such means can a living be made. If the doctor comes across an illness which may interest him, had he the time and were he reasonably paid to treat it, he cannot do so, but simply refers the patient to the nearest hospital; insurance committees do not encourage academic excursions by general practitioners.

—*Dr. F. S. Taylor-Thomas*

WHY FOLLOW ENGLAND?

Mortality rates from 32 of the 42 most common causes of death were higher (in 1934 when comparison was made) in England and Wales, which have had state medicine since 1912, than in the United States under free medicine. The general mortality rate for all causes is lower in the United States and the rate of decrease is more rapid in this country.

—*Frederick L. Hoffman, insurance statistician*

SMALL CHANGE

The expenditure of $850,000,000 for public health does not frighten business.

—*Charles W. Taussig, President, American Molasses Co., designated*

quired to support them. Experience abroad has shown that, in some of the systems at least, two employees are necessary for every physician who renders service. Since the money available for medical service and supplies barely covers the necessary cost of such service, expenditures for administrative overhead must either reduce the quantity or quality of attention or else demand larger contributions by the workers.

Moreover, when the cut was made, it obviously had to be taken from the money available for the physician and for the hospital. Obviously, this money in itself was not more than enough to cover the costs of the service if, indeed, it did that.

In 1927, a Committee on the Costs of Medical Care, composed largely of statisticians, social service workers, economists, sociologists, a few philanthropists and business leaders, and a sprinkling of physicians, was established.

A $1,000,000 STUDY

Now it is recognized that the leadership and control of this Committee was primarily that of E. A. Filene of Boston who had been for years committed to substitutes for the American system and whose views inclined toward the cooperative system as the only method of distributing goods and services. After five years in which it spent $1,000,000, contributed by various foundations in studies of medical service, the Committee made two reports. A majority recommended reorganization of medical service on an organized basis with medical care centering around hospitals in which physicians would be employed full-time on salaries to render special services, the general practitioners serving merely as feeders for the hospitals. In each state, a law administration was to be set up to control medical service. Payment was to be made on a voluntary insurance basis but there was a clear understanding that the voluntary insurance system must eventually be transmuted into a compulsory system.

The minority opposed such a revolution in medical service, insisting that medicine was undergoing gradual evolution with the introduction of new technics and that only scientific experimentation under controlled conditions could yield evidence of value as to what methods might be best in various parts of the country. They opposed particularly any breakdown of the personal relationship between doctor and patient and mutual responsibility.

Now, during the years, the advances of medical science had gradually led to lengthening of life. A child born in the United States in 1890 could reasonably expect to live about 40 years, whereas a child born in the United States today can reasonably expect to live 62 years. In 1890, 2.7 per cent of our population were more than 65 years old. Today that figure is 7.8 per cent. The increases of the aged and the onset of economic difficulties threw the problem of care of the aged into politics. Advisory committees were set up to develop technics for meeting this problem as well as unemployment and the distribution of medical service. The technical advisory committees recommended to the President the social security system for all three services, compulsory deductions from wages, taxes on employers, and contribution by the State.

by the Interdepartmental Committee as a spokesman for business before the National Health Conference. (Mr. Taussig employed Rex Tugwell when Tugwell retired from the Washington scene)

FOUR HOURS IN A CLINIC

I came at 12:30 for the afternoon clinic, and was still on my bench two hours later. Whenever I walked to the desk I was ordered to "sit down." The room was jammed, and I wondered if people around me felt as exasperated as I did at waiting. But they seemed not to mind. They looked beaten, dulled. "You'll get used to coming to clinics," said a woman next to me.

Finally my name was bawled out and a clerk pointed to an alcove. There another clerk put me through several sheets of routine questions, made out several colored cards, and didn't raise her eyes toward me during the whole proceeding. She droned out that I should go to the second floor for a diagnosis, and there, on a bench with a dozen others, I sat for another two hours.

—*Edna B. Mann in The Modern Monthly*

There was, however, a distinction as to the manner in which benefits would be administered. Under the system for old age and unemployment it was decided to give cash benefits in return for the government levies on the workers' wages, while in the case of health insurance, medical service only was to be rendered. Payment then would be made to the physician and to the hospital by the State rather than directly to the patient or to the insured worker. Thus the physician would become, not an employee of the worker, but an employee of the State.

DOCTOR'S SPOKESMEN IGNORED

The House of Delegates of the American Medical Association met in special session and explained its objections to the system and these objections were presented to the Administration. An advisory committee of 12 physicians was set up as soon as it was recognized that the so-called technical advisory committee, which had worked out the plan of compulsory sickness insurance, was not adequately representative of the medical profession. At no time has there been suitable consultation between the Administration and the medical profession as represented by the chosen spokesmen of 117,000 physicians who constitute the American Medical Association. Strangely, the powers that be invariably have chosen to consult small groups, representing frequently only a few hundred doctors, from whom, however, they were certain to get the kind of a reply that they wanted.

Meantime, the President had appointed a committee of 12 physicians and that committee opposed compulsory sickness insurance. For this reason the recommendation of the President with respect to Social Security—or "economic security" as it was first called—did not include such a departure and the law was passed without it. Instead, under Title 6, an appropriation of $10,000,000 was provided for the United States Public Health Service and the Children's Bureau of the Department of Labor under the system of grants-in-aid with matching appropriations by the states. That system sets a sinister pattern for paternalistic federal legislation in purely state matters. It includes acceptance of federal dictation because the state cannot get the money unless the federal Government approves its plans.

It has been said that the federal Government has never rejected a plan of a state, yet it must also be recorded that the spenders in the state are not likely to bring to the federal Government a plan which is certain to be rejected because it does not coincide with the federal system. Indeed, the ultimate has been reached by the appearance before state legislatures of representatives of federal bureaus who have brought measures for passage by the state legislatures and have insisted upon the passage of these "model statutes," with possibly the change of a comma or a semicolon, in order that the political aims of the bureaucrats might be achieved.

Federal spending for new health schemes did not end with Title 6 of the Social Security Act. Other measures have been passed, including the LaFollette-Bulwinkle act with the appropriation of $3,000,000 the first year, $5,000,000 the second year, $7,000,000 and up after the third year for a campaign on venereal disease. This

PROGRAM FOR A HEALTHY GOVERNMENT

The fact is that the advocates of head patting and nose wiping want, or at least are willing to accept, a socialized nation as the price of paternalism. The battle with them, therefore, ought to be waged on that front. Health is not the issue; democracy is. As a matter of fact, the prime public problem in the United States today is not at all the health of the citizen; the prime problem is the decidedly poor health that the federal government is enjoying. The latter is obese with bureaucracy and ravenously hungry for more taxes to satisfy its self-generated, insatiable appetite. It ought to be put on a programmatic diet before it eats us out of house and home. Is there a doctor in the house?

—*Journal of the American Medical Association*

money likewise is spent under the technic of grants-in-aid with matching appropriations. There is also an appropriation of $750,000 a year for five years for the establishment of a national institute for the study of cancer, and other appropriations for research throughout the nation.

When measures for changing the complexion of the Supreme Court and for reorganizing the federal Government were introduced in Congress a considerable portion of the nation rose in revolt. The result was, of course that Congress failed to approve these procedures. In the meantime, the little group of serious workers which had stimulated the majority report of the Committee on the Costs of Medical Care and which had tried to plant compulsory sickness insurance into the Social Security Law had not surrendered their hopes and ambitions. The value of health promotion as a political asset had not lost its magic.

This was the time when it began to be a little difficult for idle W.P.A. hands to find something to do. The medical profession began to hear of a national health survey. It was planned to spend about $4,000,000 and employ 6,000 W.P.A. workers to make a house to house canvass of 700,000 urban families and 50,000 rural families with a view to determining the amount of chronic disabling illness and the extent to which it received medical attention.

A RICKETY FOUNDATION

Out of this national health survey came later a mass of statistical evidence. It was poorly prepared, as might have been expected from 6,000 W.P.A. workers without medical training and suddenly impressed into the highly technical task of assembling medical data for statistical analysis. The areas investigated, it is believed, did not represent a fair cross-section of the popuation. They were heavily overweighted by the large urban centers. The attempt to extend the figures so compiled naturally produced questionable conclusions. Nevertheless, on this foundation was to be based the most audacious plan of federal spending yet proposed. The great drive for government health insurance, or State medicine, was on.

Soon there was announced the appointment by the President, with an appropriation by Congress, of the Interdepartmental Committee to Coordinate the Health and Welfare Activities of the United States Government. Miss Josephine Roche, third assistant secretary of the Treasury, in charge of the United States Public Health Service, was designated as Chairman of the new committee. Promptly a technical advisory committee was named. It included Isidore Fallk, formerly a worker for the Committee on the Costs of Medical Care—later associated with John Kingsbury, an employee of the Millbank Fund, in his drive for compulsory sickness insurance—and still later an employee of the Social Security Board which has been empowered to study medical care. He particularly believes that compulsory sickness insurance is the only possible answer to the problem of medical care for the people.

About this time, rumors began to circulate to the effect that Relief Administrator Harry Hopkins was carrying about in a portfolio a Ten-Year Plan for the nation's health. Rumors were widespread to the effect that an economic conference had been held in

REDUCING STATISTICS TO FACTS

There remain only 13 counties in the whole of the United States, no part of which is within 30 miles of a general hospital, and eight of these counties have a population of less than five per square mile.

Assuming that in those counties partly within and partly without hospital areas, one-half of the population lives at a distance greater than 30 miles from the nearest hospital, we find that there are in those counties, 1,828,735 people so situated. The population in those 13 counties numbers only 67,800. Adding these two figures, we find that 1,896,535, or roughly 1,900,000 persons, live 30 miles or more from a general hospital—1.5 per cent of our total population. In other words, 98.5 per cent of the people already have, as far as geographical distribution is concerned, hospital facilities sufficiently readily available.

—Rev. Alphonse M. Schwitalla, President, Catholic Hospital Association

TAKE IT AND LIKE IT

A meeting has just been held at Washington on the subject "Children in a Democracy." . . . the handling of the meeting itself was undemocratic. A pile of documents was presented to the audience on the education of children, their health, and kindred subjects. The pile stood almost a foot high. The meeting lasted a week. Newspaper accounts represented that this pile of documents was "discussed" by the delegates. The public was led to believe that the results were spontaneous As a matter of fact, the whole program was framed weeks in advance, the material was prepared by those who had their own special cause to plead, and no chance was given for all who might disagree with the points made to prepare and present a contrary view. I do not mean that this conference was something that should be criticized. It was a good thing. But it was not democratic and it did not deal with children in a democracy. It offered to an audience assembled in Washington certain conclusions by a few persons who had worked on certain problems, and the audience was asked to think as its leaders thought, and they did so. The decision was the only one they were offered an opportunity to make.

—Dr. Terry M. Townsend, President, Medical Society of the State of New York

which one group had recommended the spending of $4,000,000,000 to aid industry and agriculture by pump priming and another group wanted to lay out the same sum on a health pump. It seemed to them that, in the good name of health, it would be possible to drain swamps, build roads, construct hospitals and diagnostic institutions, and spend vastly for supplies and equipment.

The medical profession, through its headquarters in the American Medical Association, tried repeatedly to gain some information as to the nature of the proposed national health program. Important senators and other officials were besought for light as to its content. Every one of them confessed, some with embarrassment, that the existence of the document was known but that it had been absolutely impossible for them to obtain the slightest reference to it. Everything was held back as a mystery to be sprung at the National Health Conference.

HOW TO RUN A CONFERENCE

Among the remarkable manifestations of our time is the development of the conference as a means of propaganda and promotion. Once men of knowledge, recognized as experts in their field, used to be assembled so that the information and judgment of all could be applied to the solution of serious problems. Soon it became apparent that the trend of thought and the conclusions of such a group might easily be directed toward a certain end, provided those responsible for the assemblage exercised proper astuteness in preparing the program and selecting those who were to attend. The ultimate efficiency was finally attained when the conclusions began to be drawn before the conference assembled, with the certainty that only those conclusions could be the outcome of the entire performance. Such was the National Health Conference which met in Washington in 1938.

When the National Health Conference was called, it was announced that, for the first time, the National Health Program would have a public unveiling. Of those invited to attend, 80 per cent, it was known in advance, would agree to the conclusions that were to be offered. The other 20 per cent were a grudging concession to democracy.

The program on the first morning was carefully devoted to a series of addresses attacking the medical profession of this country as a group primarily concerned with its own income, callous toward illness, oblivious to human suffering, and unaware of either the nature of the problem of medical care or of any procedure by which a solution could be sought. The addresses had been assembled, mimeographed and released to the press in advance.

On the second day, the National Health Conference began really to function. By this time it was discovered that even the seating had been carefully planned, so that no two of the opposition could sit side by side and give each other aid and comfort. Also observers were admitted to the gallery only with tickets issued by Miss Roche and her assistants. For the first time, on the basis of the fallacious statistics of the National Health Survey, it was proposed to increase federal appropriations for health from a current expenditure approximating $135,000,000 annually for all medical purposes to

THOSE DOLLAR OPTIMISTS

Neglected illness is not always convertible by means of money grants or administrative measures into illness effectively prevented or cared for. Sincere enthusiasts who 30 years ago were sure that tuberculosis would be abolished by 1935 are still writing optimistic tuberculosis programs in glamorous terms of fresh millions of dollars.

—Dr. S. S. Goldwater, Commissioner of Hospitals, New York City

SPEAKING FROM EXPERIENCE

For the most part, American doctors are determinedly opposed to this drive for socialized medicine, and I must say I am with them all the way. State medicine, in my opinion, bears disaster for doctor and patient alike. You cannot pipe out medicine to the community as you do with steam heat.

—Dr. A. J. Cronin, English author of "The Citadel"

$850,000,000 a year for some specific medical purposes. Isidore Falk also proposed that a national assessment of four per cent of the pay roll be levied to provide something like $2,800,000,000 with which to encourage a nation-wide system of compulsory sickness insurance.

OPPONENTS ARE GAGGED

At the final sessions of the conference a number of hatchet men performed, all known as much for their forensic ability as for their absolutely established views in favor of the National Health Program. This section of the Conference was broadcast. It is not recorded that any of the views of any of the opponents of the plan were put on the radio.

Immediately after the National Health Conference, attempts were made to establish small groups in various cities throughout the nation with a view to setting up agencies for promoting the program and winning its adoption. The conference itself and the program were sent to the President, who in turn sent them to the Congress, with a recommendation for careful study.

While these blueprints for national health at so much a head were being drawn, other strange and related developments began to transpire in Washington. The Interdepartmental Committee was coordinating. Nearly all phases of executive department activity participated in the extraordinary drive that followed.

E. A. Filene, who had promoted the Committee on the Cost of Medical Care and the Twentieth Century Fund, was behind the promotion of a cooperative health plan, the Group Health Association, among employees of the Home Loan Bank Board and Home Owners Loan Corporation in Washington.

Group Health Association was organized as a corporation, presumably for the purpose of providing medical service and hospitalization for employees of the H.O.L.C. and their families, although the certificate of incorporation makes every civil employee of the Government eligible for membership. The contract between the Association and the H.O.L.C. still remains a secret. It is known that the Board contributed $20,000 the first year and an equal sum the second year.

Congress never has approprated any funds for this purpose. When the Comptroller General's office made an investigation at the request of a Senator it found that the $40,000 outlay was unauthorized and irregular.

Dues in the G.H.A. were first set at $2.20 a month for single memberships and $3.30 for married, without regard to size of family. A general medical and hospital service was to be provided, subject to a few exceptions. Later, these rates were jacked up radically. Now a new member must pay a $5 application fee plus $1 for each dependent, and a $10 membership fee covering all dependents. Dues are still $2.20 a month for the principal member, $1.80 for husband or wife, $2.20 for each other dependent over 21 and $1 for each dependent child under 21. This amounts to $103 the first year for a married man with three children, and thereafter $84 a year.

THERE ARE POLLS AND POLLS

In 1938 the headlines widely and variously interpreted a poll of doctors by the American Institute of Public Opinion as evidence that they were dissatisfied with the existing system of private medicine. A closer study of the poll showed that 73 per cent had merely expressed approval of the principle of "voluntary health insurance where an individual insures himself medical and hospital care by making regular payments to a health fund." The doctors interpreted the question correctly, but some of the commentators and propagandists saw in the poll support for a form of governmental control such as that proposed in the Administration's National Health Program.

A recent survey of 20,000 physicians by the medical journal, *Modern Medicine*, shows how erroneous such conclusions were. In this poll 88 per cent answered "no" to the question: "Would you cooperate with a federally administered and controlled legislative program tending toward drastic curtailment of private practice of medicine?" The 12 per cent minority opinion seems to represent the reform strength in the profession, both within and without the American Medical Association, since this poll included non-members as well as members.

THE PERFECT TRIBUTE

In the city of Milwaukee I am considered a radical by many, but, after listening to you people here, I think that I am quite a conservative.

—*Dr. John P. Koehler before the National Health Conference*

But this isn't all the cost. Members pay $1 for the first house call in any illness. Subsequent calls "made at the instance of the attending physican" will not be charged for. The qualification is significant. For any chronic ills during the first three years the member must pay part of the cost. And there are other exceptions.

Group Health at first was strictly under the wing of the government agency. Supplies and equipment for the clinic were bought through the government purchasing facilities at the usual large discounts, a privilege distinctly irregular. Telegram, clerks hire and telephone tolls were charged to H.O.L.C., which likewise provided the first stock of printed forms and took care of electrical installation, repairs, etc. Even office equipment and supplies came from the same source, although some office furniture and typewriters later were paid for.

After the organization had been nursed along by the Government for about two years, the Filene foundation seems to have taken over the financal burden. Since then it has paid all administrative costs, estimated to be at least $15,000 a year. That is why the director of Group Health can claim the association is out of the red. It has never actually paid expenses, according to the most reliable information available.

CO-OP PLAN FOR MEDICINE

The American Medical Association and its constituent bodies have always opposed health plans of the G.H.A. type as unsound forms of "contract practice." They do not object to private health insurance that indemnifies the insured up to an agreed amount in cash, and they have cooperated with, even organized, many local "prepayment" plans with cash indemnity to spread the cost of emergency illness. But here was a purely cooperative scheme fostered by and subject to the control of the Government itself.

It has been pointed out that the standard Rochdale cooperative principle cannot be made to apply to the distribution of medical service. In a regular co-op for the distribution of goods the advantage held out to members is "patronage dividends." Established prices are paid for goods and the profits divided later in proportion to the amounts that members have bought. It is to the interest of both individual and group to buy as much as possible through the co-op. But in a medical cooperative the group interest is best served by members making as little use of the service as possible and thereby keeping down costs so that the full service may at all times be available. This conflicts with the individual interest, because the individual member pays his dues and uses every occasion to get his money's worth out of the membership.

(To be continued)

"GIVES INSTANT RELIEF"

And so we come here—the medical profession—called to a conference on a national health program. I leave it to you whether or not we have been called to a conference or whether the patient whom you represent has not asked the medical profession to write a prescription for Radway's Ready Relief which the patient has written and wants the medical profession to sign so he can get the prescription filled. That is not scientific medicine and that is not scientific economics.

—Dr. Morris Fishbein before the National Health Conference

THE POWER OF MONEY

I was pretty well depressed last night after this meeting. I felt as if somebody had handed me a little premature two-pound baby that was blue and cold and said "Here you are" and then put a check for $1,000,000 in my pocket and said, "Now, in one year we will have a 200-pound specimen that can lick Joe Louis."

—Dr. Clifford G. Grulee, Secretary, American Academy of Pediatrics, before the National Health Conference

HOSPITAL REFERENCE BOOK IN SPANISH

Distribution of the first hospital reference book in Spanish appeared in August 1940.

Under the title, *El Libro del Hospital Moderno,* The Modern Hospital Publishing Company, Inc., is publishing a reference book in Spanish on hospital organization, planning, administration and professional work for Latin American countries. The volume will contain more than 400 pages, including a directory of hospital products and a catalog section.

A total of 34 articles will appear in the new book. All articles are in Spanish and most of them are illustrated with floor plans and photographs.

Society Proceedings

Post Graduate Clinical Conferences

The first clinical conference will be held at the Hotel Kaskaskia, LaSalle, Illinois, on October 3rd. The meeting will start with a 12:00 o'clock luncheon and all physicians of the 2nd Councilor District are invited and urged to attend this important meeting.

The 7th Councilor District has arranged to have a clinical conference at Decatur on October 8th.

The Henry County Medical Society held a meeting on August 29th at Kewanee. Doctors H. B. Hullerman of Springfield and Arthur F. Abt and Aaron Arkin of Chicago were the speakers.

The Schuyler-Cass County Medical Societies sponsored a Heart Clinic at the Beardstown Hospital on September 4th. Doctor Robert S. Berghoff and Angelo S. Geraci of Chicago conducted the clinic.

Dr. Frank Deneen of Bloomington addressed the Madison County Medical Society on September 6th.

Dr. Carroll W. Stuart of Chicago addressed the Effingham County Medical Society at Effingham on September 10th, subject, "Diseases Around the Jaws."

Dr. William L. Brown of Chicago addressed the Bureau County Medical Society at St. Margaret's Hospital, Spring Valley, on September 10th, subject, "Radium and Its Application in Modern Therapeutics."

Dr. F. W. Merrifield of Chicago will address the doctors and dentists of Jersey-Greene County Medical Societies on September 13th, at White Hall, Illinois.

Dr. Max Cutler of Chicago will address the Coles-Cumberland County Medical Society at Mattoon on September 18th, subject, "Roentgen or Radium Therapy."

Doctors Guy Van Alstyne and Gerard Krost of Chicago have been invited to present the scientific program before Fulton County Medical Society at Canton on September 19th.

Dr. H. E. Davis of Chicago will present a scientific program on "Indications for Use of Radium and Deep X-Ray in Treatment of Carcinoma."

Marriages

DAVID A. GERSHENSON, Fairfield, Ill., to Mrs. Lucille Pomeroy, of Carmi, in Reno, Nev., May 18.

STANLEY C. USALIS, Kenilworth, Ill., to DR. MARGARET MARY VIDAS of Chicago in Windsor, Ont., Canada, July 14.

Personals

Dr. Gustavus M. Blech has been awarded the Military surgeon's medal and made an honorary member of the Mexican Association of Military Surgeons by the Mexican Ministry of National Defense.

At the annual meeting of the Illinois Psychiatric Society the following officers were elected for the coming year:

Dr. Charles F. Read, President.
Dr. Clarence A. Neymann, Vice-President.
Dr. Eugene I. Falstein, Secretary-Treasurer.
Dr. Percival Bailey, Councilor.
Dr. James C. Hassall, Councilor.

Dr. Max Thorek has been made an Honorary Fellow of the Surgical Academy of Peru, South America.

Dr. Charles S. Woods, Cleveland, has been appointed general superintendent of the Methodist Hospital, Peoria.

Dr. I. Pat Bronstein, Chicago, discussed "Endocrine Disturbances in Childhood" before the Madison County Medical Society in Madison, June 7.

Dr. Adrien H. Verbrugghen, Chicago, lectured on "Intractable Pain and Its Management" before the Kane County Medical Society in Geneva, June 12.

Dr. Roy S. Bothwell, Batavia, has been elected to emeritus membership in the Illinois State Medical Society, having completed fifty years in the practice of medicine.

Dr. Melvin R. Guttman, Chicago, addressed the McHenry County Medical Society, July 25, on "Diagnosis and Treatment of Middle Ear Complications."

Dr. Charles F. Reed, Elgin, was elected president of the Illinois Psychiatric Society at its annual meeting recently; Dr. Clarence A. Neymann, Chicago, (was named vice-president and Dr. Eugene I. Falstein, Chicago, secretary-treasurer.

Dr. George A. Wiltrakis, assistant managing officer and medical director of the Chicago State Hospital, has been appointed managing officer of the Alton State Hospital. He succeeds Dr. Felix W. Sokolowski, resigned. Dr. Edward Ross has been acting officer.

Dr. and Mrs. Stanley R. Walker, Chebanse, were entertained at a banquet, June 2, to celebrate their golden wedding anniversary and Dr. Walker's completion of fifty-one years in medical practice. Dr. Edwin S. Hamilton, Kankakee, was toastmaster.

Dr. W. A. Newman Dorland has been appointed by the president and trustee of the University of Pennsylvania the accredited rep-

resentative of his class at the Bicentennial Celebration Ceremonies in Philadelphia on September 20 and 21.

Dr. Benjamin Goldberg of Chicago has been elected president-elect of the American College of Chest physicians at their annual meeting, held at New York City, June 8-10, 1940. Paul H. Holinger of Chicago was elected secretary-treasurer of the College.

News of State

—The American College of Chest Physicians has opened offices at Chicago on July 1, 1940, and they are now located at 500 North Dearborn Street. The Physicians Postgraduate Press, publishers of "Diseases of the Chest" and "Diseases of the Nervous System," have their business offices at the above address.

—The 19th annual scientific and clinical session of the American Congress of Physical Therapy will be held September 2, 3, 4, 5 an 6, 1940, at the Hotel Statler, Cleveland, Ohio.

—The 6th annual meeting of the Mississippi Valley Medical Society will be held at the Hotel Fort Armstrong, Rock Island, Illinois, September 25-26-27, 1940.

—The Children's Memorial Hospital will conduct a four weeks' graduate course in pediatrics, beginning September 30. The fee for the four weeks' course is $100. Registration should be made at least one month before entrance if possible and should be accompanied by a $10 deposit. This will apply on the fee for the course and will be refunded if the applicant is unable to participate. Inquiries should be addressed to Graduate Course, 707 Fullerton Avenue.

—Dr. Edward A. Piszczek has been temporarily appointed head of a new county health service for unincorporated areas and villages in Cook County which have no public health facilities. According to the newspapers, July 19, the appointment was believed to be for sixty days, when a civil service examination is expected to be held to select a permanent head of the service. The new service was created in June with county and state funds.

—The state health department will present two new exhibits in its annual health exposition at the Illinois State Fair in Springfield, August 17-25, in cooperation with the Public Service Company of Northern Illinois. One will depict the hazards of night driving and the inability of the eye to cope with these hazards. Dioramas, transparencies, working models, apparatus, street and highway lighting units and the scientific equipment used in research on the subject will be shown. The other exhibit shows 2,000 years of progress in the evolution of resuscitation from drowning, gas asphyxiation and electric shock. Dr. Hart E. Fisher, Chicago, chief surgeon of the public service company, will be in charge. Other features of the health department's exposition will be free blood tests, medical examination of children, exhibits on "contact" diseases and nutrition. A motion picture program will be presented daily.

—New quarters for the department of psychiatry of the Juvenile Detention Home with expanded services in the treatment of these cases have been installed on the third floor of the court building at Ogden Avenue and Roosevelt Road, newspapers report. The new arrangement, under the direction of Dr. Paul L. Schroeder, state criminologist, will provide seven private offices for consultation with the youths brought in for mental examination, a staff office, ample space for clerical workers and records and waiting rooms. Instead of a large public waiting room, the waiting space has been divided into cubicles each accommodating four persons. Entrance to consultation offices will be private. All children will be given a physical examination in the court building instead of being taken to their neighborhood clinics as heretofore, making possible a complete medical and psychiatric report within twenty-four hours.

—State district health officers and nurses held a special conference on poliomyelitis recently in Springfield at the call of Dr. Albert C. Baxter, director of the Illinois Department of Public Health. Drs. Sidney O. Levinson and Philip Lewin, Chicago, addressed the meeting. Dr. Levinson discussed treatment, with special emphasis on measures for prevention of crippling. Dr. Lewin described the work of the National Foundation for Infantile Paralysis and also discussed after-care of patients, including methods of splinting and physical therapy. District health officers will be prepared to assist physicians throughout the state in diagnosis and

treatment, it was announced. Each district health superintendent will have at his office a supply of convalescent human serum and emergency splints so that prompt treatment may be instituted in the home as soon as the diagnosis is established.

Deaths

JAMES STEWART ARCHIBALD, Danville, Ill.; St. Louis University School of Medicine, 1908; a Fellow A.M.A.; served during the World War; on the staff of St. Elizabeth's Hospital; formerly on the staff of the Decatur and Macon County Hospital, Decatur; aged 57; died, July 17, of coronary thrombosis.

WILBER PRICE ARMSTRONG, SR., Springfield, Ill.; Homeopathic Hospital College, Cleveland, 1884; member of the Illinois State Medical Society; aged 79; died, July 7, of pulmonary edema, diabetes mellitus and arteriosclerosis.

ISRAEL BAIKOVICH, Chicago; Chicago Medical School, 1915; aged 65; died, July 8, in the Mother Cabrini Hospital of carcinoma of the prostate.

GEORGE STUART BOWER, Galesburg, Ill.; Northwestern University Medical School, Chicago, 1894; member of the Illinois State Medical Society; past president and secretary of the Knox County Medical Society; for many years county coroner; on the staff of the Galesburg Cottage Hospital; aged 71; died, July 14, of coronary thrombosis and hypertension.

JAMES ALLEN COTTON, Peoria, Ill. (licensed in Illinois in 1899); aged 71; died, June 10, in the Methodist Hospital of bronchogenic carcinoma of the lung.

CHRISTIAN HERMAN DIEHL, Wood River, Ill.; St. Louis College of Physicians and Surgeons, 1908; a Fellow, A.M.A.; for many years district health superintendent for the Illinois State Department of Public Health, and health officer of Roxana; formerly managing officer of the Lincoln (Ill.) State School and Colony; on the associate staff of the Alton (Ill.) Memorial Hospital; aged 60; died, July 17, in St. Joseph's Hospital, Alton, of hypertensive heart disease.

PHILIP H. FEIGEN, Chicago; Chicago College of Medicine and Surgery, 1907; aged 67; on the staffs of St. Mary of Nazareth Hospital and the Michael Reese Hospital, where he died, July 25, of coronary thrombosis.

TAYLOR WILSON FUNKHOUSER, Danville, Ill.; Rush Medical College, Chicago, 1916; a Fellow, A.M.A.; served during the World War; aged 50; on the staffs of St. Elizabeth Hospital and the Lakeview Hospital, where he died, July 18, of carcinoma.

PHILIP C. GILTNER, MAUNIE, Ill.; University of Louisville (Ky.) Medical Department, 1889; member of the Illinois State Medical Society; aged 73; died, June 27, of a self-inflicted bullet wound.

FREDERICK CLARENCE HANMORE, Chicago; Bennett College of Eclectic Medicine and Surgery, Chicago, 1906; member of the Illinois State Medical Society; police surgeon; aged 57; died, June 17, in the Wesley Memorial Hospital of pulmonary embolism and lung abscess.

JOHN A. HAUSER, West Salem, Ill.; Eclectic Medical Institute, Cincinnati, 1882; also a pharmacist; aged 81; died, June 28, in Quincy of uremia and hypertrophy of the prostate.

JOHN HUND, Peoria, Ill.; College of Physicians and Surgeons of Chicago, 1887; aged 86; died, June 23, in St. Francis Hospital.

TITUS SAMUEL LAPP, Waynesville, Ill.; Northwestern University Medical School, Chicago, 1931; a Fellow, A.M.A.; aged 39; died, July 11, in the Deaconess Hospital, Lincoln, of a gunshot wound received when he was cleaning his gun.

FELIX JOSEPH LOWNIK, Chicago; Bennett College of Eclectic Medicine and Surgery, Chicago, 1915; a Fellow, A.M.A.; aged 46; died, July 11, of cerebral hemorrhage.

WILLIAM L. MCBRIEN, Staunton, Ill.; St. Louis College of Physicians and Surgeons, 1898; on the staff of St. Francis Hospital, Litchfield; aged 75; died, July 10, of carcinoma of the bladder.

THOMAS J. O'MALLEY, Chicago; College of Physicians and Surgeons of Chicago, 1896; member of the Illinois State Medical Society; served during the World War; formerly member of the board of education; died, June 19, of myocarditis.

WILLIAM KEPLER PARKER, Dix, Ill.; College of Physicians and Surgeons, Keokuk, Iowa, 1883; member of the Illinois State Mediral Society; aged 89; died, June 7.

JOHN DUSTIN POLLARD, Chicago; Bennett College of Eclectic Medicine and Surgery, Chicago, 1915; aged 70; died, June 23, in Lake Geneva, Wis.

AUGUSTUS RALF REDER, Aurora, Ill.; Washington University School of Medicine, St. Louis, 1894; a Fellow, A.M.A.; aged 68, died, July 9, in Hot Springs National Park, Ark., of carcinoma of the liver.

JOSEPH ADRIAN STEFANSKI, Chicago; Hering Medical College, Chicago, 1909; member of the Illinois State Medical Society; on the staff of the South Chicago Community Hospital; aged 55; died, July 3, of pneumonia.

CLARK LEROY WARREN, Hines, Ill.; Rush Medical College, Chicago, 1897; aged 69; died, June 25, in the Veterans Administration Facility of tuberculosis.

EDGAR MORGAN WEBSTER, Chicago; Starling Medical College, Columbus, 1884; formerly on the staff of the South Chicago Community Hospital; aged 80; died, May 9.

ANTHONY G. WITTMAN, Elgin, Ill.; Northwestern University Medical School, Chicago, 1905; member of the Illinois State Medical Society; veteran of the Spanish-American War; assistant managing officer of the Elgin State Hospital; aged 64; died, July 16, of bronchogenic carcinoma.